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VOL. 23, NO. 6 JUNE, 1955

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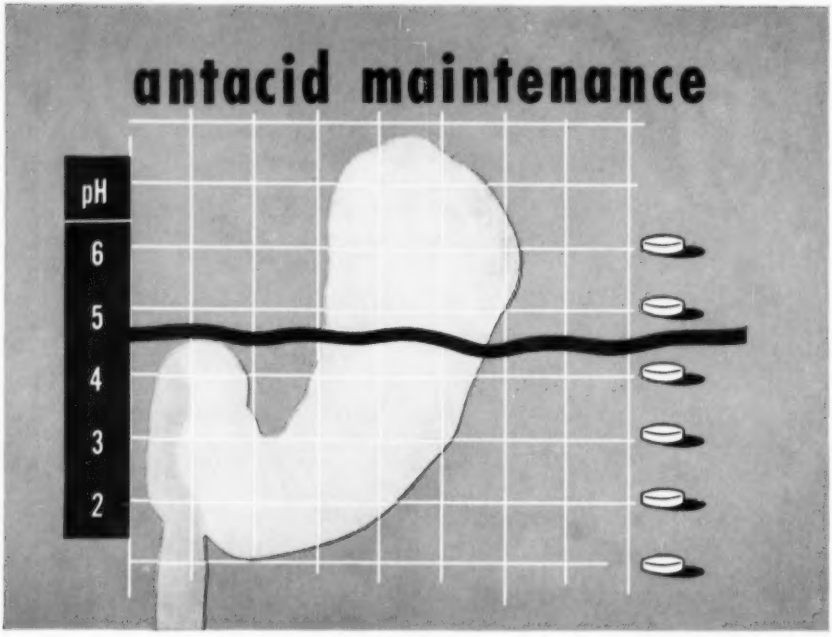

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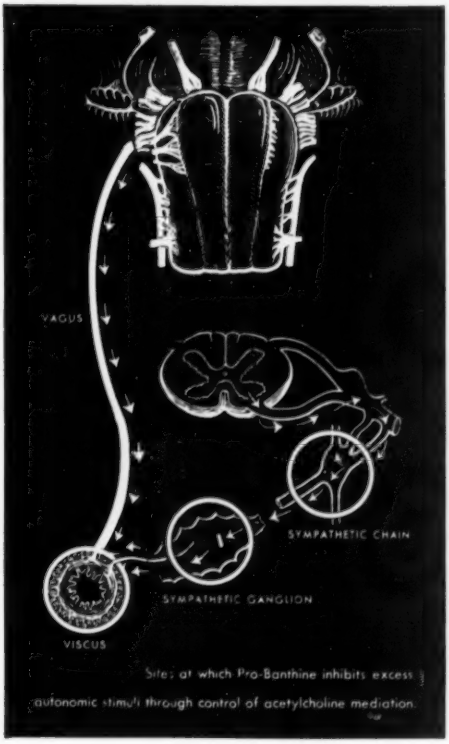

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1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25*:416 (Nov.) 1953.
2. Roback, R. A., and Beal, J. M.: Gastroenterology *25*:24 (Sept.) 1953.

SEARLE

VOL. 23, NO. 6 JUNE, 1955

THE American Journal OF Gastroenterology

(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

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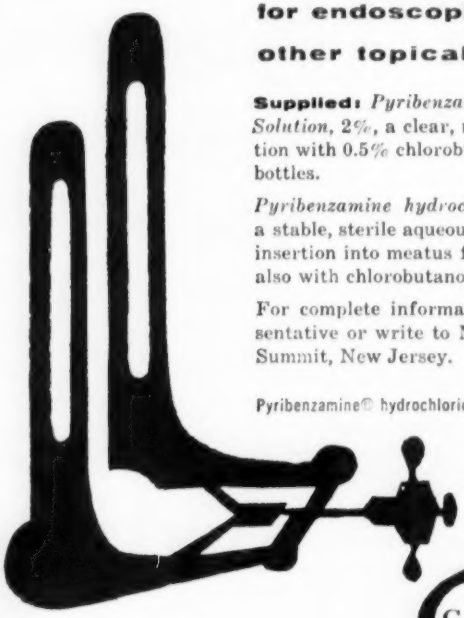

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PANEL DISCUSSION ON ESOPHAGEAL VARICES*

IRVING B. BRICK, M.D.
EDDY D. PALMER, Lt. Col., M.C.
CHARLES A. HUFNAGEL, M.D.
and
EDWARD J. JAHNKE, Jr., Maj., M.C.
Washington, D. C.

Dr. Brick:—About three years ago Dr. Palmer and I began an investigation, primarily by means of esophagoscopy, of all patients with cirrhosis who entered several hospitals with which we were associated, including the Walter Reed Army Hospital and the V.A. Hospital here in Washington. In this study, routine esophagoscopy was done in each and every cirrhotic proved by biopsy, whether or not the patient had bleeding. It was our feeling that in this manner a good many patients who had not bled from varices with cirrhosis would be discovered to have varices.

The reason that we were particularly interested in the problem of esophageal varices is and was that it now constitutes the main cause of death from gastrointestinal hemorrhage. Obviously, the major cause for entrance into the hospital for bleeding is peptic ulcer, primarily duodenal ulcer, and we found that the main cause of death was bleeding from the esophageal varices.

We are going to show more on the diagnosis of esophageal varices, subsequently, by Dr. Palmer, but these tables will give a bird's-eye view into the problem as we found it, and to the various points that we found.

The management of bleeding peptic ulcer, because of the combined medical and surgical forces, usual in a hospital, is such that deaths from peptic ulcer are far less than they are from varices. Table I shows the results of a study in a moderate-sized hospital.

*Presented before the First Annual Convention of the American College of Gastroenterology, Washington, D. C., 25, 26, 27 October 1954.

In the five-year period there were 18 deaths from esophageal varices due to cirrhosis, whereas in the same time there were only six deaths from bleeding from peptic ulcer, both duodenal and gastric. It is interesting to note that three times more deaths occurred from esophageal varices bleeding than from peptic ulcer bleeding. The statistics have been verified in a much larger study from Cook County Hospital, Chicago, where in a study of 600 such deaths it was found that esophageal varices proved the largest in the cause of death.

TABLE I

DEATHS DUE TO GASTROINTESTINAL HEMORRHAGE

	Tot. Aut.	Cirr. Var.	D.U.	G.U.
1947	137	2	1	
1948	108	4		
1949	111	1		
1950	134	4		
1951	186	7	2	3
	676	18	3	3

At the Cleveland City Hospital, Simeone reported 77 per cent fatalities in 68 cases of bleeding varices. Table II shows the results of the first 238 cases of cirrhosis that were examined. The series is somewhat larger at this time. You will note, however, that in the 238 cases in this table, varices were found in more than half, 161. You will note, too, that there were 87 cases in which varices were found without a history of hemorrhage. This is an important

TABLE II

INCIDENCE OF ESOPHAGEAL VARICES
DETERMINED ESOPHAGOSCOPICALLY IN CASES OF CIRRHOSIS

	Cases
Total	238
Esophageal Varices	161
Esophageal Varices and Hemorrhage	74
Esophageal Varices with no Hemorrhage	87

group in this problem, and will be discussed later, as to the management of the patient who has esophageal varices but has had no hemorrhage.

Table III briefly shows in the first 147 cases the unsatisfactory status of x-ray examination. You will note that by esophagoscopy in this first small group, 92 patients were diagnosed as having varices, whereas by x-ray only 19 patients had a positive diagnosis. This topic will also be discussed somewhat further.

Table IV reveals an attempt to correlate some of the physical findings with patients having cirrhosis, and it is broken down into patients with varices

and patients without varices. You will note that the only notable difference appears to be in the finding of spiders. It has been our impression that when a patient with cirrhosis has vascular spiders, the chances of his having varices seem to be increased.

With this introduction of the problem, I am going to now ask Dr. Palmer to discuss particularly diagnosis, and anything else that comes into his mind.

TABLE III

Comparison of X-ray and Endoscopy (147 cases)

Esophagoscopy		X-ray	
		Varices	No Varices
Varices	(92)	19	73
No Varices	(55)	0	55

Dr. Palmer:—In our experience esophagoscopic examination is very much more effective than radiologic examination in the detection of varices. We have found that in only about 20 per cent of those who have varices seen by esophagoscopy, does the radiologist find signs that permit him to make the diagnosis.

Of course, when one makes a statement such as this he is not damning radiologic technic or the role the radiologist plays in the study of the patient with varices. This is especially true, of course, if there has been hemorrhage

TABLE IV

Esophagoscopic and Clinical Findings in 229 Cases of Cirrhosis

Esophagoscopy	Varices present	Varices absent
No. patients	154	75
Hepatomegaly	116 (75.3%)	68 (90.6%)
Ascites	47 (30.5%)	16 (21.3%)
Spiders	91 (59.0%)	23 (30.6%)
Splenomegaly	52 (33.7%)	18 (24.0%)

from the gastrointestinal tract, because cirrhotics may have bleeding lesions other than varices. As we all know, there seems to be a statistically significant increase in the incidence of peptic ulcer in the cirrhotic patient.

The responsibility of the clinician, in the face of massive hemorrhage from a known cirrhotic, is first, to refrain from automatically assuming that varices are at fault, and to recall that this patient, more than the next, is likely to have

a peptic ulcer. Also he should be aware of the fact that erosive gastritis is an extremely common cause of hemorrhage in the patient who happens to have esophageal varices also.

I think that we have to recognize that, as with so many other phases of medicine, technical progress in the surgical attack on varices has far outstripped understanding of the pathophysiology of portal hypertension and varices. I bring this up because we have been so discouraged in trying to predict ahead of time the presence or absence of esophageal varices after making a physical examination. We have found no correlation, or practically none, between the presence of varices and the findings upon physical examination.

In most parts of the country a diagnosis of esophageal varices is based upon the physical examination plus the appearances on x-ray examination, and yet the experience in Washington is that neither approach gives valid information. We have found no correlation between the presence of varices and the size of the liver, the presence of ascites, splenomegaly, or prominent abdominal venous pattern. The only correlation we have been able to make, and a very interesting one, is one between the presence of spiders and varices. This is particularly interesting because, of the physical findings, the presence of spiders appears to be the one least directly associated with the level of the portal venous pressure.

Dr. Brick:—What Dr. Palmer has pointed out emphasizes something that we have come to realize in the routine study of the cirrhotic, and that is that before a cirrhotic bleeds, if you happen to see him at that stage of the game, it is well to do an upper gastrointestinal series. That is true in all of these patients, and we also believe that all of these patients should be at least esophagoscoped, and preferably have esophagoscopy and gastroscopy, because of the apparent increase in the appearance of upper gastrointestinal lesions in cirrhotics, and particularly in the cirrhotic who has portal hypertension.

This problem therefore seems to divide itself into two phases: the patient who has varices and has no hemorrhage; and the patient who comes into the hospital bleeding.

I wonder if Dr. Hufnagel will tell us about the approach to the acute problem of bleeding in the patient who has a definite diagnosis of esophageal varices.

Dr. Hufnagel:—It is worthwhile to recall that the massive hemorrhage from esophageal varices has implications beyond those of actual blood loss. It is not infrequent for us to see a patient who is admitted after an exsanguinating type of hemorrhage, who has immediate blood replacement and cessation of bleeding, by one of the methods we will mention, who regains normal pulse, blood pressure, and hemoglobin, but who goes into a coma and succumbs with

no further blood loss within the next 28 or 48 hours, after he is once seen in the hospital.

This tendency of the liver to be massively damaged by either the hypoxia of acute hemorrhage or the sequellae thereof, certainly is something which we must not forget in the consideration of the overall treatment of the patient, and particularly in relation to prevention of the first episode of bleeding, which may be in itself fatal in a high percentage of cases.

Probably the simplest and the most useful method for the control of acute esophageal bleeding is the use of the Sengstaken-Blakemore type tube, a double-balloon tube—which allows one to aspirate the stomach contents, while tamponading the stomach and esophagus. It is again well worthwhile to know there are many variants of this type of tamponade, and one can obtain, under most circumstances, very reproducible and good results. One of the important things is that the gastric balloon of the tube be filled quite full, and if one inflates it with 500 to 600 c.c. of air and puts a moderate amount of traction on it, actually most bleeding will be controlled by this without any inflation of the esophageal balloon at all. Obviously, it is through a communication between the portal system in the abdomen and the esophageal veins which come through the esophagus itself or the cardia of the stomach, or the esophageal tissues that pressure in this area by good firm tamponade can reduce the tension in the esophageal varices. If the prothrombin time of the patient is within relatively normal limits, and blood clotting factors are not abnormal, one can obtain cessation of bleeding often by this method alone. The inflation of the esophageal balloon gives one an indication of cessation of bleeding. If it is not immediately effective, then blood is regurgitated.

A gastric balloon, first inflated and pulled up and followed by packing of the esophagus has been used by some for the clotting of varices. It is cumbersome but sometimes useful in the patient who is comatose and acutely ill and in whom it may be difficult to pass the Blakemore-type tube. The small tube may be passed fairly regularly, under esophagoscopy, if necessary.

The use of acute ligation procedures for the control of bleeding has been rarely necessary in our experience, and I think that Dr. Jahnke has something to say about that. We would prefer, in most instances, if we can bring the hemoglobin levels to relatively near normal, control bleeding abnormalities, and then do an emergency shunt procedure.

Dr. Brick:—Before Dr. Jahnke takes over, I should like to say one thing about the acute bleeder. With the management that has been outlined by Dr. Hufnagel, generally that suffices. Unfortunately, however, we do encounter situations in which that procedure does not suffice, and the patient keeps bleeding. Dr. Palmer has had some experience with the use of injections of esophageal varices during the acute phase. Will you tell us something about that, and also something about the efficacy of that method?

Dr. Palmer:—It has been our habit to be mighty cautious about accepting varices as the source of bleeding when the average cirrhotic comes in with hemorrhage, as I have said. It has therefore been our usual procedure to carry out esophagoscopy, gastroscopy, and roentgen study immediately after the patient has been admitted to the hospital, as soon as the blood has been started, and so forth.

This has given us a chance to use sclerosing treatment for bleeding varices, and I think that it is fair to say that sclerosis of the actively hemorrhaging varix has a little bit to add to the efficacy of the Blakemore tube. As Dr. Hufnagel has said, we must rely on pneumatic tamponade, and certainly sclerosing is no substitute.

If we can find the point of the varix that is actually bleeding, and this is possible during hemorrhage much more often than you might think, we put a needle into the lumen of the varix on the other side of the bleeding point. We take the opportunity to measure the portal pressure for later reference, feeling that knowledge of the level of the portal pressure will be of help to the surgeon later on. With the needle still in the varix, we inject sodium morrhuate. We use 3 c.c. in the bleeding varix, pick up two others in the neighborhood and inject 3 c.c. into each of these. Before this is carried out, the Sengstaken tube is put in place and the gastric balloon blown up. I think it would be quite a mistake and perhaps not very useful to inject sclerosing solution if one did not have the instruments for tamponade immediately available. As soon as the injections have been made, we place tension on the gastric balloon and blow up the esophageal balloon, and hope that that increases the possibility of varix sclerosis.

As you all know, sclerosing therapy of esophageal varices was begun back in 1939, in Scandinavia, but it was rather quickly abandoned because it was not possible for the pathologist to prove that sclerosis was actually being accomplished. Possibly by the addition of tamponade we can encourage sclerosis and help prevent the possibility of immediate recurrence of hemorrhage.

You have to remember, of course, that whenever you sclerose a varix, you are adding to the burden on the portal bed and increasing portal hypertension. This can never be considered good definitive therapy.

Dr. Brick:—This brings us to the point of discussing the most definitive therapy we have at the present time. Obviously, this, too, is not the ultimate answer to the problem, in our opinion; however, in our hands, and in the hands of many others, it does seem that the surgical technics for decompression have a great deal to offer. There is no dispute about patients who have varices and who have bled, as to the efficacy of and the necessity for such surgery.

Dr. Jahnke, will now discuss this particular aspect of the problem and, before he goes into the surgical procedure and technic, I should like him also

to tell us when, after a bleeding episode, is he willing to undertake this type of surgery. By "when" I mean what condition does he want the patient to be in, particularly, because that does seem to be an important fact in the minds of many.

Dr. Jahnke:—When considering the application of the shunt operations in the definitive treatment of portal hypertension, there are four important questions which must be answered in each case.

First, what type of shunt will be most effective in the individual patient?

Second, what operative mortality can be anticipated?

Third, what alterations in liver function will occur during and after the operation?

Fourth—and probably of greatest importance to the patient—just how effective is the portal decompression:

There have been numerous operations devised for the relief of portal hypertension. The only ones that have withstood the test of successful clinical application have been the portacaval shunt and the splenorenal shunt. Of these, we prefer the direct portacaval shunt since we believe this particular operation will produce the greatest degree of portal decompression.

Clinically it is often difficult to differentiate an intrahepatic from an extrahepatic portal obstruction. Since the surgical approach to each differs, it is essential to have an accurate visualization of the portal vascular system prior to operation if a useless procedure is to be avoided. This data can be obtained by the routine utilization of the technic of percutaneous portal venography. A #18 gauge spinal needle is inserted into the spleen through the left lateral chest wall. Approximately 30 c.c. of contrast material are rapidly injected and a roentgenogram of the upper abdomen is taken just at the completion of the injection. Examination of the picture obtained will indicate the site of block in the portal system and will also indicate the largest portal tributary, be it splenic, superior mesenteric or portal vein, that can be utilized in the shunting procedure. It will also indicate any severe degree of angulation that may occur by the utilization of any of the aforementioned vessels. On the basis of the splenogram, the following types of shunts have been established: portacaval, splenorenal, splenocaval and superior mesenteric-caval. In each instance, the procedure performed represented the largest venous bypass possible.

This brings one to the second question which must be answered; namely, what mortality rate can be anticipated? The patients prognosis once bleeding has occurred is most grave. Approximately 50 per cent will succumb to their first episode of hematemesis and as high as 70 per cent will be dead within one year. Thus an operative mortality rate of less than 20 per cent has been

considered tolerable. We have found that even this need not be anticipated if certain precautions are taken. With the avoidance of anticoagulant therapy, hepatic failure represents the major cause of death. Liver function surveys were performed daily during the pre- and postoperative periods and revealed two factors which were significant in substantially lowering both the morbidity and mortality rates. Intensive medical therapy should be initiated and operations should be delayed until the hepatic surveys indicate stabilization of the liver. The degree of abnormality is of little significance as long as the level of function is the maximum possible and remains constant.

The second important factor has been the routine use of intravenous chlortetracycline during the postoperative period. It should be given in daily doses of one gram until the liver function has returned to the preoperative level or has become stabilized at some other level. The important point to remember is stabilization. Surgery is delayed until it occurs and similarly chlortetracycline is administered until it returns.

By adhering to these dictums we have lowered our mortality rate to 6.6 per cent. On this basis, we have also extended the scope of the operative procedure and now recommend it as a prophylactic measure for patients who have portal hypertension and esophageal varices who have yet to experience their first, but possibly fatal, hemorrhage.

The third question that requires a satisfactory answer before one can safely recommend the shunt operations is, what alterations in liver function can be anticipated following operation. Serial liver function studies performed daily during the postoperative period have revealed a direct correlation between the degree of operative and anesthetic trauma to which the patient is subjected and the degree and extent of early post shunt hepatic dysfunction. An operative procedure which takes two hours to perform will result in mild hepatic alterations which will return to normal in two or three days. To extend the operating time to six hours will result in marked changes in liver function which may take three weeks or more to return to the preoperative level. Graded but similar changes occur in the times between two and six hours. Similar curves have been observed depending upon whether intravenous chlortetracycline was or was not given during the postoperative period. Greater alterations occurred in those patients who did not receive the antibiotic.

Interval liver function surveys have been performed up to two years after operation. In the majority of patients we have found no deterioration in the hepatic function beyond that which could be normally anticipated in the cirrhotic individual. In fact, many patients have shown slight improvement, especially in the total protein, serum albumin and A/G ratio determinations. In contradistinction to the experimental work in which portal bypass caused marked deterioration in the liver, this has not been observed in the clinical patient.

The answer to the fourth question, how effective is the portal decompression, forcefully demonstrates the true value of the shunt operation. Several tests have proved to be extremely valuable in arriving at this conclusion. The first test is the rectum to lung ether circulation time. Two or 3 c.c. of ether are vaporized to 200 c.c. and are injected rapidly into the upper rectum through an inlying rectal tube. The time from injection till recognition on the breath is measured and has been found to be proportionate to the portal pressure. The upper limit of normal in the adult male is 25 seconds. It is slightly less in the adult female. In our cases, the preoperative portal circulation times have ranged between 26 and 68 seconds. During the early and late post shunt period these times have been within normal limits, the range being 11 to 26 seconds. This was objective evidence as to the function of the venous bypass.

The second test used was the actual manometric measurement of portal pressure change at the operating table. The upper limit of normal is considered to be 120 mm. of saline. In all patients the preshunt pressures varied between 250 and 560 mm. of saline. Following decompression, the average drop in pressure was over 200 mm. of saline. In every instance, the final pressure reading was below 300 mm. of saline. This is the level, below which, postoperative esophageal bleeding is considered unlikely.

The third test was esophagoscopic examination of the varices with direct varix pressure measurements when possible. Following decompression the varices disappeared in 65 per cent of patients. In 20 per cent they were collapsed and no pressure readings could be obtained. In the remaining 15 per cent varices were seen, however, they were smaller than those noted before operation and were under considerably less pressure as determined by direct measurement. During the late follow-up, a composite graph of the three previously described tests showed good correlation with each other and gave definite evidence as to the patency of the shunt. In several patients who had splenorenal shunts performed and who had subsequent episodes of hematemesis, closure of the anastomosis could be determined by the tests. There was an increase in the portal circulation time, and increase in the direct varix pressure measurement and an increase in the size of the varices. In patients with adequately functioning portacaval shunt, these determinations remained unchanged for periods of months and years depending on the length of follow-up.

The final test for determination of shunt patency was to repeat the percutaneous splenogram. If the bypass was functioning the dye could be followed through the portal vein, through the shunt and into the inferior vena cava in the direction of the heart. This test could be utilized only in those patients who had no previous splenectomy.

Of greatest interest to the patient and of greatest clinical significance to the physician was the fact that only four patients had any evidence of bleeding following operation, an incidence of 7.5 per cent. In each case the hemorrhage

was minor. Two of the patients have since gone over 2 years without further recurrence. An even more graphic demonstration is possible. During the preoperative period, the 53 patients required 178,750 c.c. of whole blood to combat their numerous episodes of bleeding. Postoperatively, the same group of patients required only 2,500 c.c. of whole blood.

In conclusion, the effect of the shunt operation has been studied in 53 patients who had portal hypertension and esophageal varices. The results obtained in this group would seem to indicate that the procedure is of definite value once hemorrhage has occurred. It also demonstrates that the operation might safely be recommended in those patients with portal hypertension and associated varices who have yet to experience their first, but possibly fatal, episode of bleeding.

Dr. Brick:—It is a rather dramatic statement such as this that indicates to all of us the efficacy, by and large, of portal decompression in this group. Our experience has certainly been along the line that after the portal decompression, the episodes of bleeding, if they do occur, are modified, and in many cases it has been indeed dramatic to see, esophagoscopically, the varices disappear. After all, that is the crux of the matter: does the operation cause a diminution in the size of the varices? Case after case has shown that.

I wonder if Dr. Hufnagel has anything to say on the subject of surgery, and also I want him to say a few words on the use of portal decompression in the emergency case, that is, doing a portacaval shunt or splenorenal shunt in the patient actively bleeding, and in whom the methods we have talked about, pneumatic tamponade, and possibly injection of the varices, have not been successful in stopping the flow of blood from the varices.

Dr. Hufnagel:—Dr. Jahnke has, I think, given you a very excellent summary of the status of the shunting procedures. We have felt very strongly that shunting procedures offer the best type of operative intervention for the relief of portal hypertension, and we will employ them electively over any of the other procedures which have been suggested from time to time.

As you know, there are vogues in all things. Medical vogues appear from time to time for the use of procedures in which the splenic artery and the hepatic artery, etc., are ligated, in preference to doing some shunting operation. The theories behind this are somewhat at variance, and we employ them only in unusual circumstances.

The other type of procedure, which is sometimes of real help, is the use of resection of the upper third of the stomach, and the lower third, to half of the esophagus. This, I think, should be reserved primarily for patients in whom shunting operations have, for one reason or another, failed, and this occasionally does happen, under the best of circumstances.

I assume you are all familiar with the fact that resection of the esophagus and the stomach leaves one with a situation which is far from ideal in terms of the living conditions of the patient. We certainly try in all cases to avoid that type of esophagogastrectomy procedure, but it surely can be extremely helpful in the massive bleeder who is not controllable by other shunting procedures.

I think that Dr. Brick's question about the emergency use of shunting operations has been answered to some degree by what Dr. Jahnke said earlier in his comment, but I think it is important to point out that one does not enter upon such an emergency type of shunting operation without some degree of preparation. The situation usually arises somewhat in this fashion. The bleeding can be stopped with the Blakemore tube, but as soon as one removes the tube, in a short period the bleeding recurs. Under those circumstances the patient often is in good general condition—he has been under observation and control, so he does not have a great hemoglobin depletion. In that case I would elect to control the bleeding and go ahead and do the shunt as an elective procedure rather than doing anything else, but I think it is foolhardy to attempt to do any shunting procedure of a major type in a patient whose general status is highly unsatisfactory.

There is one thing which we must do in all types of surgery at this time, and that is really adequately prepare the patient properly whether it be for elective or emergency procedure. The overall mortality from the shunting operation, in patients who have been selected well and handled well preoperatively, has greatly decreased, and shunting is a procedure that can be used as an elective where the patient has not bled.

We have been extremely fortunate. We have not lost any patient who was operated on prior to his first bleeding episode, and I think that this indicates, too, that the safety of the procedure in in a large degree related directly to the way the patient is prepared and the status of the patient prior to operation.

Dr. Brick:—In this study we have been distressed by the incidence of patients who die from their first hemorrhage. We will have these patients in the hospital and we will have the work-up in progress. In some cases it is decided not to do anything because they have not bled and then, out of a clear sky, actually, and literally under our very eyes, they have their first hemorrhage and die. This problem has made us well aware of the fact that sometimes we are doing routine esophagoscopies in these patients and discover varices before bleeding. We have had to face the problem of what to do about the patient who has varices and who has not bled. I would like an opinion on this from Dr. Palmer at this time.

Dr. Palmer:—We feel very strongly here in Washington—and I think we are all in agreement on the point—that one should not let the patient prove that he is able to bleed once in order to meet the criteria for this type of surgery.

Of the patients that we have been able to study, who have been shown by direct examination actually to be bleeding from varices, 50 per cent have died during their first hemorrhage, and approximately 50 per cent, again, with the second hemorrhage, and third hemorrhage, etc.

We have taken a lot of stock in the statistics of Dr. Patek and others as to the probability of hemorrhage in the cirrhotic patient. We realize that hospital experience may be misleading when it comes to estimating the danger of hemorrhage in the cirrhotic patient with varices. We see patients when they bleed and not so often when they don't. Nevertheless, our experience agrees with that which has been recorded in the literature from several centers. In general the chances of hemorrhage are so great and each hemorrhage is so dangerous that no further argument is needed for going ahead with surgical portal decompression as soon as varices are discovered.

Of course, anyone who recommends or carries out surgical portal decompression is assuming several serious responsibilities, not the least of which stems from the fact that we know very little of the natural history of portal hypertension and varices. In about 10 per cent of the patients we have studied, varices have disappeared spontaneously and apparently permanently. We don't know why. We are caught in the dilemma of recommending a dangerous surgical procedure in a desperately ill patient, whose varices might disappear spontaneously as time goes on. We need more information to guide us in our approach toward varices, but for the moment, we think we are right in assuming a radical attitude in regard to surgical therapy.

Dr. Brick:—Another factor is the very definite fact, in my opinion, that when you see a patient with cirrhosis who bleeds, and you transfuse him, and the bleeding stops, it is more harmful than just the bleeding. When you start studying the mortality figures of deaths from bleeding varices, in many cases you don't take into consideration the fact that these patients may recover from bleeding only to go into coma; only to have ascites or other signs of decompensation in the days and weeks that follow. This has been impressed upon us quite emphatically in the patients that we have observed.

They do very badly when they bleed, and having seen these patients die in their first hemorrhage, we feel that this group of doctors, at least, ought to consider the future program for the cirrhotic with varices who has not bled, and also consider portal decompression.

We have now done approximately 30 such cases. It is also true, and I am sure the surgeons will agree that most of the patients with cirrhosis and varices, who have not bled, are, by and large, in better condition than are the patients who have bled.

I want to ask Dr. Hufnagel a further question. Where do these portal decompression procedures fail? What is the mechanism by which it happens, so that their varices don't disappear? I would like you to state your ideas on that.

Dr. Hufnagel:—These are really fairly simple. Possibly the shunt which is established is small because of anatomical reasons which may be present. The end-to-side shunt should, under most circumstances, be a highly effective one. One has to be extremely careful about the positioning of the splenic vein. In the postcaval shunt, one has to be careful about the size of the incision, but we rarely get a failure in an end-to-side portacaval shunt. If it does fail, it is again probably due to angulation of the vessel caused by excessive trauma at the time the operation was carried out, or due to perhaps some disease of the wall of the portal vein which was a problem at the beginning.

All of these things, most of which are of a technical nature, are reasons for failure of shunts. When one does a side-to-side shunt, this type of anastomosis tends to fail unless one does particular things such as excising parts of the walls of both vessels involved, and being careful to make a very adequate anastomosis.

Dr. Brick:—We have discussed primarily the patient with cirrhosis who has varices. It is true that in our series of cases as well as in other series of cases, there is a smaller group in which cirrhosis is not the problem. I wonder if Dr. Jahnke would discuss that particular group of patients where cirrhosis is not the problem causing portal hypertension.

Dr. Jahnke:—We are a little unhappy when we find a patient with an extrahepatic block because the results of treatment, namely the splenorenal shunt, have been far from satisfactory. In this regard, however, the percutaneous portal splenogram has been of value. It has the advantage of demonstrating all the large venous channels associated with the portal circulation. The best one of these can then be chosen for use in the shunting procedure. In one patient in whom a splenorenal shunt would have been the procedure employed, the splenogram revealed a circulatory configuration which would make a splenocaval shunt technically more satisfactory and with much less angulation of the involved vessels. The splenic vein was mobilized, rotated behind the root of the mesentery and anastomosed, end-to-side, into the inferior vena cava. The result was most satisfactory.

In other instances the splenogram has revealed a large superior mesenteric vein which would allow a shunt of greater area than using the splenic vein. In these instances a side-to-side anastomosis was performed. Again, the results have been quite satisfactory.

In the main, the results of splenorenal shunt for extrahepatic portal blocks have been so poor that we make a determined effort in all cases to find a more satisfactory vessel to use in establishing the venous shunt.

Dr. Brick:—It is our experience that when the patients have varices secondary to something other than cirrhosis, the prognosis is much better. Will Dr. Palmer discuss this very definite group, without definite cirrhosis, which we

have encountered, because in each of the cases done in this particular series, liver biopsy has been performed, and we are occasionally faced with the problem of the biopsy not showing cirrhosis and definite varices being present.

Dr. Palmer:—I suppose the world over the commonest cause for esophageal varices is schistosomiasis. Second on the list is cirrhosis, and third, extrahepatic portal block.

There are other causes of portal hypertension, and these are of extreme interest to all physicians. First of all, in the second half of pregnancy we frequently find esophageal varices, and these may be rather extensive in distribution. We have examined several cases by esophagoscopy, and in some the varices have been very large. We have seen no pregnant woman in whom the varices have bled. Surely the explanation for portal hypertension in pregnancy must be some sort of mechanical infringement on the portal system, secondary to the enlarging uterus.

Another interesting group has been patients with heart failure. In one half of the patients with chronic heart failure, whom we have examined, there have been varices.

Might I interject an explanation here and say that we don't go around esophagoscoping pregnant women and people with heart failure, to find out what is down in their esophagi. We have been called upon to make examinations in cases of foreign body, in cases of obstructed deglutition, stricture, etc., and we have been surprised to find a high incidence of varices, especially when we weren't particularly on the lookout for them.

To get back to heart failure, a very interesting facet of the picture in the patient with heart failure is that, unlike other situations, the varices are largest in, and at times are limited to, the middle third of the esophagus. We have no explanation for this, anatomically.

We have seen severe hemorrhage from varices in patients with heart failure. Emergency therapy of such patients is a very difficult problem. In the group of cardiac patients with varices which I refer to, liver biopsy has shown chronic passive congestion, some portal infiltration, and a degree of portal fibrosis, but no perilobular fibrosis. So we were not dealing with actual cirrhosis.

Perhaps the most significant group of all for us to keep our eyes on are patients with acute infectious hepatitis. We have been surprised and excited to find that, when it has been necessary to esophagoscope patients with acute infectious hepatitis, in about half there were varices. We have gone through the centuries in medicine assuming that esophageal varices are passive shunting devices which require a long time to enlarge in response to an increase in portal pressure. But, although we have found the varices in acute hepatitis to be small, we have found them early in the course of the disease.

Dr. Brick:—Dr. Palmer has touched on the fact that possibly some doctors will criticize the widespread use of esophagoscopy in various types of patients, such as pregnant women, hepatitis, and congestive heart failure. Occasionally this group becomes symptomatic and requires esophagoscopy of necessity. In our experience, with the semi-flexible esophagoscope, in many hundreds of cases, there have not been any instances, even when varices have been present, which resulted in any bleeding. This is a question that is frequently asked: what is the danger of bleeding in esophagoscopy if varices are present, and in our hands, this combined group of these patients do not seem to have had any difficulty, even in many patients when we have had to push the scope out of the way of the varices to see them. We have seen no bleeding.

An interesting thing in some few of the cases of esophageal varices, has been the presence of dysphagia. We have had several cases in which that has been primary, and when you see an almost complete obliteration of the lumen, you wonder why we don't get dysphagia as a symptom more frequently in the presence of esophageal varices.

I think, unless any other member of the panel has some other comment to make on this subject, we would be willing, Mr. Chairman, to entertain any questions that the group here might have.

Questions and Answers

Dr. Charles C. Teresi (Rochester, N. Y.):—Have there been any culture studies and studies on material by liver biopsy to show the existence of bacteriological matter?

Dr. Brick:—Dr. Sborov, in his series at Walter Reed did do that very thing. They did take liver biopsies and culture them aerobically and otherwise, and it is my impression that the results of such bacteriological study were rather negative. Is that correct Dr. Jahnke?

Dr. Jahnke:—We tried initially to determine the possible site of action of intravenous aureomycin in the patients with portal hypertension. It was based on previous studies which showed that, in patients with bacterial endocarditis, the concentration of bacteria was greater in the portal vein than in the hepatic vein. This suggested that the liver might act as a filter. Biopsies of the liver were taken at laparotomy in over 50 patients, many of whom had liver disease. Both aerobic and anaerobic cultures were performed. In only two instances was any growth observed. One was an Aerobacter aerogenes and the other a fungus. On the whole, the results were negative.

Dr. C. Wilmer Wirts (Philadelphia, Pa.):—I will take the opportunity at this moment to interject that we likewise have performed bacteriological study on liver biopsies in thirty cases, and found two positive cultures. We did not feel this significant; one was probably a contaminant.

Dr. Edward Levy (New York, N. Y.):—In the case of hepatic ligations do you do those culture studies?

Dr. Jahnke:—No, sir.

Dr. Brick:—Our experience with hepatic ligation has been so discouraging in the few cases that we have had the opportunity to observe, that, in the first place, we never did advocate it, and never have done it. Some of the surgeons who did, soon turned their patients over to us, and most of them died. Of the three at Georgetown, two died, and at Walter Reed it has been minimal, but I think it has been as discouraging an experience as ours.

Dr. Jahnke:—That is right.

Dr. Brick:—We didn't do bacteriological studies in those patients, though.

Dr. Donald W. Mitchell (Washington, D. C.):—With the use of the inflatable tubes is there any criteria about the length of time they should be left in? I know of three cases of necrosis of the esophagus, and one of rupture, from the use of these tubes over a period of time.

Dr. Palmer:—That is a very important point, Dr. Mitchell. We have made it a practice to leave balloons in place only for three days. We have been in the habit of deflating the balloons with the tubes still in place for a few hours, just to be sure we won't have to put them back immediately. So far it would appear that three days is a fairly safe period for retention of the balloons, even when considerable pressure is being exerted on the esophagus, and I think there would not be much point in increasing the sojourn of the balloons so far as preventing hemorrhage is concerned.

Dr. Brick:—We have not been impressed by the inflation of the esophageal balloon and, as Dr. Hufnagel pointed out, we think more important is the pressure of the balloon that goes in the stomach, plus the actual putting of pressure through a traction apparatus.

I agree that two to three days is the maximum we dare keep the balloons in place, and I think you will find that the patients who have had necrosis and, as you point out, perforation, probably have had their balloons in longer. Have you any information on that? How long did they have them in?

Dr. Mitchell:—Two of them, in one hospital, had them in for seven days, and the one that had the rupture was actually ruptured from too energetic pumping of the esophageal portion of the balloon.

Dr. Wirts:—Which type of balloon was used?

Dr. Mitchell:—The Blakemore.

Dr. Wirts:—Dr. Palmer, which type of balloon do you prefer?

Dr. Palmer:—We think the Sengstaken modification of the balloon is the best.

Dr. Donovan C. Browne (New Orleans, La.):—I would like to have your evaluation of the hepatic vein and venule catheterization for determination of portal hypertension in differentiating extrahepatic and intrahepatic blocks, comparing it with splenic injections for direct portal bed visualization.

Dr. Brick:—We personally have had no experience with that at Georgetown.

Dr. Palmer:—We have had it just experimentally.

Dr. Brick:—Do you have an answer to Dr. Browne's very interesting question?

Dr. Palmer:—I suppose, from what I have read of the other two methods, all three give rather valid results, Dr. Browne. I know you will say so if you don't agree. I am just basing my answer on what I have read. The proper answer would depend then on which procedure is safest, quickest and easiest on the patient. I would think that a measurement of pressure within the esophageal varices would be quickest, and I also think it would be safest. I don't know how uncomfortable the other two methods might be.

SURGICAL TREATMENT OF PORTAL HYPERTENSION*

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The problems of chronic portal hypertension essentially resolve themselves into those of bleeding and chronic intractable wasting ascites. The development of esophageal varices is a part of the natural development of collateral channels to circumvent the area of portal obstruction. Portal obstruction may occur either as intrahepatic obstruction, such as in cirrhosis, or less commonly as extrahepatic block due to thrombosis of the portal vein with or without cavernous transformation of this channel. It is interesting to note that some patients who have serious block due either to intra- or extrahepatic obstruction may develop other natural channels for communication between the portal and systemic venous systems. The other natural shunts may vary in size from those which are of very small caliber to channels which may be one-half to three-quarters of an inch in diameter. The larger channels frequently run from the *porta hepatis* to reach systemic circulation by way of the anterior abdominal wall. Patients with such large shunts frequently do not manifest evidence of bleeding from the esophageal varices or hemorrhoidal veins which represent another of the primary collateral channels until something occurs which obstructs this naturally occurring shunt.

When intrinsic liver disease is present from a process such as cirrhosis or posthepatic liver fibrosis and regeneration, the problem of ascites in the chronic process is usually controllable by medical management. It is only in the extreme cases in which this result is not obtainable. Thus bleeding from esophageal varices is one of the major serious complications of portal hypertension.

Bleeding from esophageal varices is not only dangerous from the point of view of actual blood loss which in itself may be fatal, if not controlled, but hemorrhage may be the precipitating cause of liver failure due to a greatly reduced total oxygen carrying ability and liver anoxia superimposed on the already damaged liver. In addition, the large amount of blood which may be broken down in the gastrointestinal tract may produce sufficient retention of metabolites to be also fatal. Thus, it is quite apparent that serious esophageal bleeding presents major problems in addition to blood loss per se. The percentage of patients who die in the initial episode of massive esophageal bleeding is rather high, approximately 25 per cent. These are individuals primarily who have bled massively prior to entry into the hospital and in whom coma ensues shortly after admission, or in whom the bleeding is of such severity that the patient is exsanguinated on admission, or in whom superimposed liver failure rapidly ensues.

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It is also noteworthy that once esophageal bleeding has occurred it almost always recurs, and usually the episodes recur with gradually increasing frequency and severity. These facts make it highly desirable to ascertain the presence or absence of esophageal varices in all patients who have suspected portal vein obstruction of intra- or extrahepatic origin. For, as will be pointed out later in the discussion, under certain conditions it is desirable to institute treatment prior to the first episode of bleeding. The surgical treatment of portal hypertension resolves itself to two phases: first, that of the treatment of the acute bleeding episode in which primary efforts are devoted to the control of hemorrhage and to the elimination of blood from the gastrointestinal tract. In addition to control of this bleeding in the acute phase, the treatment of liver failure is mandatory.

It should be remembered in the control of acute bleeding that while the varices which produce bleeding are most often located in the lower portion of the esophagus, it is not unusual for very large veins to be present in the body as well as in the cardia of the stomach. The use of a large balloon in the stomach helps to control bleeding from this source. In addition, if the bleeding is from the esophageal area, the traction on the gastric balloon tends to compress the vessels in communication with the high pressure portal area, thus reducing the pressure in the esophageal varices, to permit clotting and cessation of bleeding.

In all cases of bleeding from the esophagus it is of extreme importance to have an immediate determination of the prothrombin time. The bleeding tendency may be greatly exaggerated by a lowered prothrombin time. This should be corrected by the usual measures, using Vitamin K oxide intravenously and transfusion. We are concerned here primarily with the surgical aspects of this treatment once the diagnosis of bleeding from esophageal varices is made; it is our policy to employ the Blakemore-Sengstaken tube which has been most helpful in the majority of cases. Prior to the insertion of the tube the balloon should be calibrated in the terms of the volume of air which is necessary to distend the balloons to a given size. The tube is then passed into the stomach and the gastric balloon inflated. We believe that it is quite helpful if the gastric balloon is distended with 300 to 500 c.c. of air. This gives a large balloon which when traction is added to the nasal end of the tube, gives a large bearing surface at the cardia of the stomach. A considerable number of cases can be controlled merely by traction on the gastric balloon, which if inflated with a large amount of air, will not pull out of the stomach. The esophageal balloon is then inflated and connected to a manometer, maintaining pressures of 40 to 50 mm. of mercury. The aneroid type of manometer is satisfactory for this purpose. When the pressure is considered adequate the tube is clamped and the balloon is left in place. The use of a rubber block or direct traction can then be applied in cases in which the simple application of the tube is not adequate. It has been our general experience that the use of the tube will stop bleeding immediately in the vast majority of cases. The gastric suction is then applied and one can

readily ascertain whether or not bleeding is occurring by inspection of the return of the gastric tube. In some patients who are irrational and semicomatose, it is very difficult to pass the large Blakemore-Sengstaken tube. In such patients a similar type of tube can be constructed which may be passed directly through the esophagoscope. Another method which has been employed by others and ourselves is to pass an Abbott-Rawsen tube into the stomach after the addition of a balloon over the proximal openings of the tube. The balloon is then inflated inside the stomach and traction is applied. The esophagoscope is then inserted into the esophagus, the esophagus is packed with gelfoam. This is a much more complicated procedure and is not as effective, in general, as the use of the Blakemore tube. If bleeding occurs on deflation of the esophageal balloon it is sometimes helpful to allow the patient to swallow small amounts of buffered thrombin and re-inflate the balloon. This promotes clotting of the eroded varix and by using one or a combination of these methods almost all acute bleeding can be controlled.

If these measures all fail, one must make the decision as to whether an immediate shunting operation is feasible and compatible with the patient's condition, or whether direct intervention upon the esophageal varices is more desirable. Direct obliteration of the esophageal varices may be accomplished by exposing the lower portion of the esophagus in the chest, opening the esophagus longitudinally and plicating the esophageal varices with longitudinal stitches. This closure of the esophagus is then carried out with or without packing of the periesophageal area. This type of therapy is, in general, useful only in the acute phase and one must elect at a later stage to do a shunt. If the patient's condition is reasonably satisfactory and the bleeding is recurrent or cannot be controlled, a shunt should be considered seriously and carried out as an emergency procedure. In all instances adequate blood replacement is of vital importance, and in no case should operation be carried out until the patient has had adequate blood replacement therapy. A situation which most often confronts one is that acute bleeding can be controlled readily with the tube, but each time the balloon is deflated or the tube is removed, the bleeding recurs in an extremely short interval. In such instances the patient's general condition has usually been maintained well by replacement therapy during the bleeding episode, and in the interval in which bleeding has been stopped by the use of the balloon, the hematocrit and other blood values have been restored to relatively normal levels. In such instances we have found it extremely useful to proceed with the immediate shunting operation when it has been demonstrated that bleeding has recurred.

Many forms of direct operative intervention have been advocated for the control of portal hypertension. It should be remembered that these have as their primary object the prevention of bleeding from the esophageal varices, and in some cases the control of wasting ascites which cannot be controlled by adequate medical management. Portal hypertension can be controlled most readily

through the use of adequate deviation of blood from the portal system into the systemic system. When interhepatic block is present the most direct and controllable method of deviation of blood is obtained through the use of a shunt between the portal vein and the vena cava, or through a shunt between the splenic and the left renal veins. We have advocated primarily the use of a direct portacaval anastomosis. This gives one a direct opportunity to establish the size of shunt which appears to be most desirable. The juxtaposition of the portal vein to the vena cava usually makes such a procedure possible without excessive angulation of either of the two veins involved. Either an end-to-side or a side-to-side shunt may be utilized. The end-to-side anastomosis tends to have certain advantages which make many surgeons prefer it. It is simpler to do and it can be done in certain patients in whom the distance between the portal vein and the inferior vena cava is such as to make impossible the establishment of a direct side-to-side anastomosis.

In addition to the shunting type of procedures which have been described, there has also been introduced the procedure of arterial ligation for the correction of portal hypertension. In general, this consists of ligation of the hepatic artery proximal or distal to the gastroduodenal branch, and/or concomitant ligation of the splenic and left gastric arteries. Another type of procedure which has been used is the direct attack on the periesophageal venous communications with section and/or packing to produce fibrosis and interruption of the collateral pathways. In addition to these measures there has also been advocated resection of the upper third of the stomach and the lower third of the esophagus with esophagogastrostomy. These latter two groups do not directly correct the portal hypertension, but have as their major aim the interruption of the communication between the portal and systemic systems. In addition, high subtotal esophagogastric resection with the concomitant vagotomy tends to reduce the acid regurgitation which may be a factor in the production of esophageal bleeding.

In the intrahepatic blocks which result in portal hypertension, the major causes are Laennec's cirrhosis, biliary cirrhosis, posthepatitic cirrhosis and more rarely the Cruveilhier-Baumgarten syndrome. In these types of disease it is important to recognize that surgical treatment is directed only at correcting the portal hypertension and that the primary pathology, that is, cirrhosis of the liver, remains unchanged. In considering surgical treatment of this type of disease the diagnosis is best established definitively by liver biopsy. This can be carried out rather simply by the needle-type biopsy. In these patients the diagnosis is usually not difficult clinically and the type of cirrhosis can be confirmed by direct examination of the tissue of the liver. The majority of these patients will not present major splenic enlargement or evidence of hypersplenism. If the spleen is greatly enlarged or if there is evidence of hypersplenism and biopsy of the liver shows an essentially normal liver, it may be presumed that in the presence of evidence of portal hypertension that the picture is not

that of intrahepatic block, but represents extrahepatic block. The percutaneous splenogram will give more evidence as to the exact site of block.

In the presence of cirrhosis the x-ray of the esophagus may present, on careful examination, evidence of esophageal varices. It is helpful and desirable in all cases to do an esophagoscopy in such patients and to measure the pressure in the esophageal varices by direct methods. This will give a direct reflection of the pressure in the portal system. Since the outlook for a patient who has had bleeding from esophageal varices is such that approximately 50 per cent will not survive for one year after the first bleeding episode, and that approximately 25 per cent will die with the first bleeding episode, we strongly feel that patients who have portal hypertension and esophageal varices with definitively elevated esophageal venous pressures should be operated upon electively before the occurrence of the first bleeding episode. We believe that a direct portacaval anastomosis is the operation of choice in such patients. An end-to-side anastomosis is, in general, the most satisfactory. We perform an end-to-side anastomosis in all patients who have a rise in portal pressure at operation of not more than 50 mm. on occluding the main portal vein. In patients in whom there is a marked rise in portal pressure on occlusion of the portal vein at operation, we will, if possible, perform a side-to-side anastomosis between the portal vein and the vena cava. It is important that an adequate stoma be established and that the size of the opening in the cava be made approximately the same as the normal portal vein without constriction. When a side-to-side anastomosis is performed, a segment of the side of the cava must be removed and the vessels must be approximated without excessive tension.

In the instances when cavernous transformation of the portal vein is present in addition to the cirrhosis of the liver, sometimes it will be possible to find a segment of the portal vein at the junction of the splenic and superior mesenteric vein which is patent and which can be utilized for an anastomosis. When, however, the distance between the cava and the patent segment is too long to make a direct anastomosis, in these patients we will use a graft of freeze-dried vein graft to bridge the gap. In all circumstances care must be taken not to twist or angulate the portal vein in such a way as to interfere with flow. Medical management of the patient preoperatively is of extreme importance, and great care is taken to bring the patient into the best possible condition prior to operation. This particularly includes restoration of hemoglobin levels to as close to normal as possible and with correction of hypoproteinemia and hypoprothrombinemia prior to operation. We attempt to stabilize the patient's liver function at its maximum level. All operations for the establishment of portacaval shunts are carried out through a thoracoabdominal approach.

In individuals in whom there is evidence of hypersplenism or in which the splenogram indicates an extrahepatic block, and in cases in which extrahepatic block could be shown by other methods, splenorenal anastomosis is the procedure

of choice. In this procedure a thoracoabdominal approach is also used on the left side and the spleen is removed. The large main splenic trunk is anastomosed end-to-side to the left renal vein. It is interesting that with increasing experience the mortality rate from both types of shunting procedures has been greatly decreased. Mortality rates vary in different clinics and may, in general, be considered in relation to the severity of the disease process and the condition of the patient's liver function. Blakemore[1] has classified his patients into good risk and poor risk groups with a mortality rate of 9.3 per cent in the good risks and 39.9 per cent in the poor risks. Linton[2] and others have reported approximately similar mortalities with splenorenal shunts. Both Blakemore and Linton have reported high mortality rates when the serum albumin level was less than 3 grams per cent. The presence of jaundice in Laennec's cirrhosis is an extremely poor prognostic sign and greatly increases operative risk. The presence of jaundice in biliary cirrhosis is of much less serious import. It has been our experience that the shunting procedures offer great relief and operative mortality in our hands has been extremely low. In the last 25 operations performed for severe portal hypertension and esophageal varices there have been no operative deaths and one late postoperative death. This includes patients in whom operation was performed because of intractable bleeding. In only one patient has there been any bleeding since operation, and all of these patients have been followed for a period of more than six months. The one patient in whom bleeding did occur, the bleeding was extremely mild and required no major therapy. The one death in this group was a late death due to liver failure. The patient who had extremely poor liver function had a side-to-side anastomosis which was patent at autopsy, but succumbed to liver failure some six weeks after operation after having made a good recovery from surgery. We, therefore, strongly feel that this operation offers patients great improvement at relatively low risks. We believe that splenorenal anastomoses tend to give a higher incidence of recurrence of bleeding, and that it is not the procedure of choice for intrahepatic block, but that it should be employed in patients where the spleen must be removed because of hypersplenism. During the postoperative period the patients are carried on a strict liver program, and in addition, they receive intravenous aureomycin, one gram per day for three to five days, and thereafter oral aureomycin for another five to seven days. It is interesting that esophagoscopy following the establishment of a shunt shows a steady and definite decrease in the size of the varices and it can be shown that these vessels become invisible by esophagoscopy in the first few months following operation. In the majority of patients direct measurement of the portal pressure at the time of operation will exceed 400 mm., and in the remainder it will exceed 200 mm. of water.

In relation to ascites it should be pointed out that rigid control of the sodium content, the diet in association with a high protein, high caloric diet will tend to control the ascites in the vast majority of patients. When ascites

is combined with bleeding a portal shunt is carried out, the ascites tends to disappear and one can ordinarily expect a good result.

The rationale for the hepatic artery ligation and the associated vessels of the portal arterial supply is based upon several assumptions, including the presence of arteriovenous communications between the hepatic arterioles and the portal venous system with concomitant increase in mortality rate following such ligations is greatly higher than that following shunts. Shunting operations have been demonstrated to be extremely safe in capable hands and the reported mortality statistics may be quoted as follows: Rienhoff[3] reported a series of 23 patients in which ten were operated on primarily for hemorrhage with a mortality rate of 50 per cent. Two of those who survived had small, but definite hemorrhages postoperatively. Kramish, Reckler and Swan[4] reported five cases of which two died, a mortality of 40 per cent. In Rienhoff's series of 23 patients his over-all mortality rate was 30.4 per cent. There has been considerable evidence that ligation of the hepatic artery in experimental animals shows an extremely high mortality rate. It is felt that a considerable factor in the death of such animals is the invasion of anaerobic bacteria, particularly gas producers, and that the greatly decreased arterial blood flow was a further factor in this invasion and in the production of liver necrosis. A considerable portion of this liver necrosis is prevented by the use of large doses of antibiotics in the early postoperative period. In the clinical cases in which operation has been utilized, however, it has been found, in general, that liver failure is common in spite of large doses of antibiotics following major ligations. It has been shown by Michels[5] that there is great variation in the arterial anatomy in the liver in man. This variation is so great that it would appear difficult to predict accurately the results of ligation at a standard point of the hepatic artery.

The revival of the procedure of resection of the stomach and esophagus for control of bleeding has produced new interest in this procedure. It is interesting to point out that in our series of cases there was one in which the portacaval anastomosis had been performed elsewhere approximately two years prior to our first seeing the patient, and following this there was evidence that the shunt had closed and that the patient had had recurrent massive attacks of esophageal bleeding. The patient then had a splenorenal shunt and again following this the shunt failed. In the twelve months following the second operation the patient was admitted to the hospital approximately once each month with bleeding of massive amounts of blood. It did not seem feasible to attempt another shunt in the face of two unsuccessful procedures. We, therefore, elected to do an esophagogastric resection with esophagogastrostomy. It has now been one year since operation and the patient has had no recurrence of his bleeding. We feel that this procedure offers an alternative to portacaval shunt in exceptional cases. We would reserve it primarily for the patient who has had unsuccessful shunts. Fortunately, these are now quite rare primarily due to improvements in the concept of the principles of the operation. Gastro-

esophageal resection carries a definite disadvantage insofar as it is necessary to place the stomach into the thorax, and this is not optimal for nonmalignant disease. We elect to do an intrathoracic vagotomy in all such patients in order to reduce the amount of acid reflux into the esophagus and the production of esophagitis. The success with shunting procedures has led us to avoid all peri-esophageal venous ligations either with or without packing, and we feel that their role is extremely limited.

Conclusion

A review of the present status of the treatment of portal hypertension has led us to the concept that with good medical management the shunting operation for the relief of bleeding from esophageal varices and intractable ascites is gaining more and more acceptance. Precipitation of major liver crises by massive bleeding is an important part of the problem of portal hypertension. Adequate direct portacaval shunt will regularly eliminate or greatly decrease this danger. In the presence of demonstrable esophageal varices with an increase in pressure in such veins we believe that it is desirable to do a portacaval shunt prior to the first episode of bleeding. A portacaval anastomosis can be expected to succeed and the mortality rate of such a procedure in the patient without extremely poor liver function can be accomplished with a mortality rate of less than 5 per cent. In the last 25 patients in which such procedures have been undertaken for the relief of portal hypertension there have been no operative or early postoperative deaths, and only one late postoperative death. This includes all cases, including acute bleeding in patients with extremely damaged liver.

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MUCOSAL PROLAPSE AT THE ESOPHAGOGASTRIC JUNCTION*

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During certain studies on the normal location of the esophagogastric mucosal junction, it has become evident that mechanical abnormalities in the region of the cardia, in addition to hiatus hernia, are not excessively rare. Redefinition of the limits of normalcy for the junction's position, necessary because of the recent observation that it may spontaneously migrate toward and away from the brink of the stomach sac[8], has focused attention on the problem of mucosal prolapse. The line of mucosal junction between esophagus and stomach may normally move proximally as far as the diaphragmatic hiatus, producing a tube-like extension of the stomach and obliterating the abdominal esophagus as such. The discrepancy between the proximal extent of the gastric mucosa and the position of organ configurational change explains why an esophagoscopically demonstrable hiatus hernia may exist at a time when the radiologist can find no abnormal luminal widening above the hiatus[9]. Clearly, many clinical problems surrounding mechanical diseases at the cardia might be dispelled by more realistic definitions of the normal anatomy.

The diseases of significance in this connection are organ intussusception and mucosal prolapse. They involve quite different anatomic and mechanical principles. Intussusception is very rare in this site[2,5,6]. Retrograde mucosal prolapse has been described only a few times in the human[3,12], although it is not a rare veterinarian problem and has even been described in the lower vertebrates[7]. The patient of Wells[12] had had esophageal obstruction for several days, caused by gastric mucosa incarcerated in the distal end of the organ. Two esophagoscopic examinations during the period of obstruction showed a fungating polypoid mass, the true nature of which was not at the time recognized. Feldman[3] reported two cases, diagnosed on the basis of the x-ray picture; his published roentgenograms demonstrate the difficulty of differentiating retrograde prolapse from small hiatus hernia.

Prolapse of the esophageal mucosa down into the cavity of the stomach has rarely been described[10,11]. Presumably it is difficult to detect and is of little clinical import. Sarasin and Hoch[11] described prograde esophagogastric prolapse under three categories: invagination associated with hiatus hernia, simple asymptomatic prolapse, and prolapse initiated by polypoid tumors. Their patient complained of intermittent swallowing difficulty.

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The present discussion considers only prolapse. During the past four years two instances of retrograde prolapse of the gastric mucosa into the distal esophagus and one of prograde esophagogastric prolapse have been recognized at this hospital. The first two were detected by esophagoscopic examination after a normal situation had been found by the radiologist, and the third could be demonstrated by x-ray only.

Case 1:—This 33-year old white man, a rather heavy drinker, was hospitalized after having vomited small amounts of blood for three days. This was the first

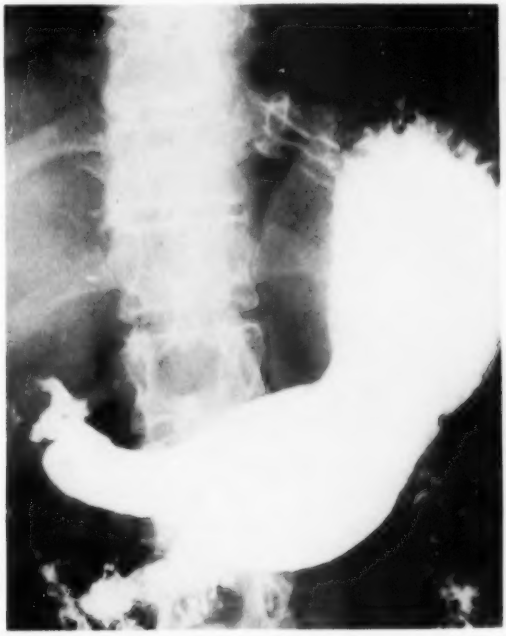

Fig. 1—(Case 1). Roentgen picture of retrograde gastroesophageal mucosal prolapse.

recognized episode of gastrointestinal bleeding, but there had been dyspepsia at intervals for three years. The latter took the form of patternless diurnal and nocturnal epigastric aching, pyrosis and occasional vomiting, appearing about four times a year and lasting about three weeks at a time. There was no relationship to food, hunger or recumbency. He had not previously sought medical help.

Physical examination, routine laboratory study and chest x-ray were normal. Stool examinations were persistently positive for occult blood. The first upper gastrointestinal x-ray examination was normal.

Esophagoscopic and gastroscopic examinations were performed under mild sedation with the patient in the left lateral position. The esophagus was normal and the esophagogastric junction in the proper position, but, as the region was watched, a strange phenomenon was observed. Each time the patient retched or gagged, the gastric mucosa appeared suddenly as a plug within the distal esophagus and slowly squeezed itself about five centimeters proximally. It could not be determined from the appearance of the mucosal plug whether this represented a complete sleeve of gastric mucosa or a fold from one segment of the cardia. The plug each time, however, could easily be pushed back into the stomach with the esophagoscope, and then it could be seen that it was a sleeve. The process of prolapse and manual reduction was repeated several times.

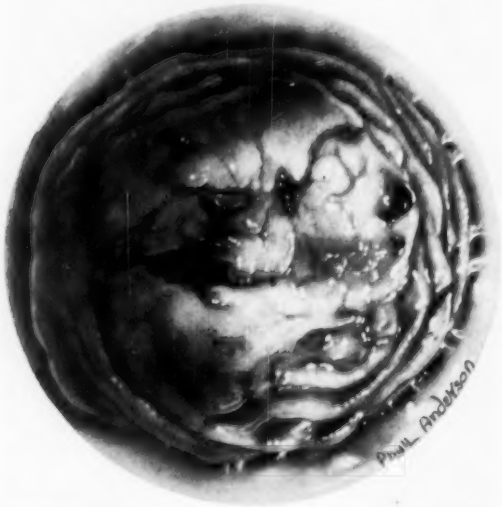

Fig. 2–(Case 2). Esophagoscopic view of prolapsed sleeve of gastric mucosa in esophageal ampulla. The endoscopic diagnosis was carcinoma, until an attempt to pass the esophagoscope beyond the tumor caused the prolapsed segment to return to its normal position.

Spontaneous reduction was not observed. The prolapsed gastric mucosa was intensely hyperemic and bled slowly. Gastroscopic examination was then done, and it was found that there was generalized erosive gastritis, with a small amount of bleeding.

Repeat roentgenologic study succeeded in demonstrating the gastroesophageal retrograde prolapse (Fig. 1). It could from time to time be produced with the Valsalva maneuver but not by the Trendelenburg position or by pressure exerted against the abdominal wall. The prolapse regularly remained until the patient was given another swallow of barium, whereupon the mucosal sleeve returned

to its normal position. Throughout the examination the patient had no unusual sensation, and his pain was not reproduced.

The patient was treated with bismuth subcarbonate and belladonna. The stools became guaiac-negative after a week. The vomiting cleared quickly. There was no improvement in the pain and pyrosis. The patient was returned to duty with the diagnoses of erosive gastritis and retrograde gastroesophageal mucosal prolapse.

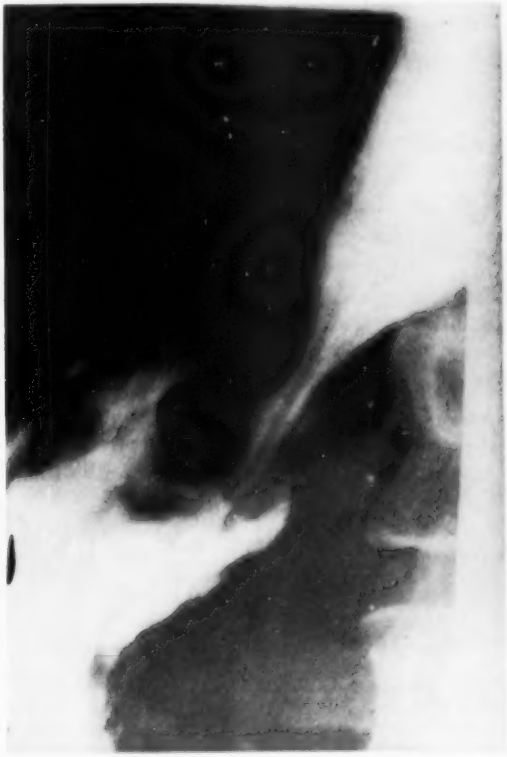

Fig. 3—(Case 3). Prograde esophagogastric mucosal prolapse, an incidental roentgen finding in this patient.

Case 2:—This 21-year old white soldier was hospitalized because of intermittent difficulty in swallowing. He had been well until two years previously, when he began to have epigastric soreness after swallowing solid food. After several months, vomiting supervened and the patient sought medical help. He reported to a large diagnostic clinic where the diagnosis of "spasm of the esophagus" was explained to him. He was treated with a brief course of dilatations with a hydrostatic dilator. Thereafter he was asymptomatic for one year,

but dysphagia, retrosternal distress and pyrosis then returned. X-ray study confirmed the diagnosis of achalasia, and he was transferred to this hospital.

Physical examination showed a young man who appeared well and who had no abnormal physical findings except for absence of the second swallowing sound upon repeated examinations. Laboratory studies were entirely normal.

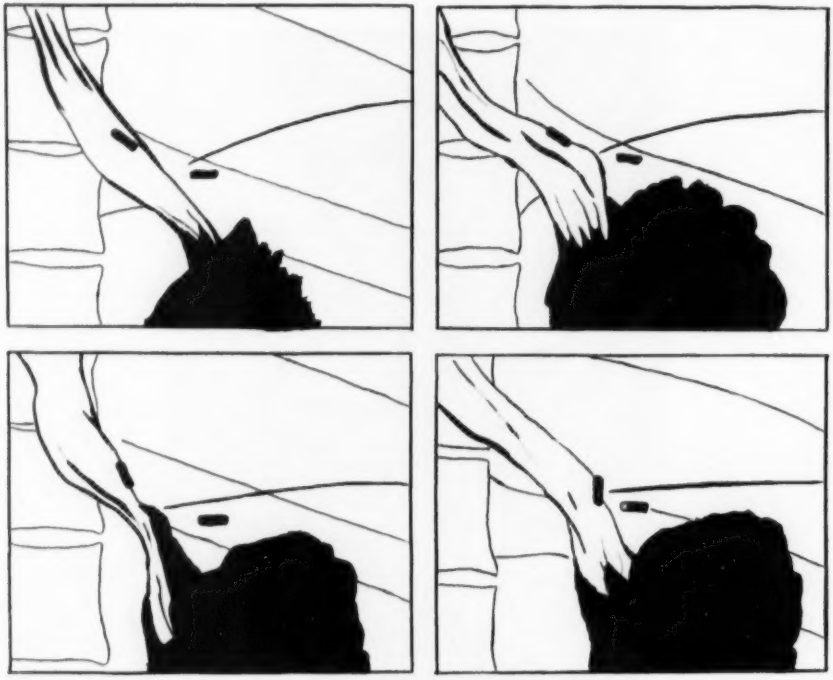

Fig. 4—Tracings of four spot-films taken at intervals over a period of about 10 minutes, each after a swallow of barium. One silver clip had been tied into the muscularis propria at the left lateral aspect of the esophagogastric junction, as the site was determined from external appearances. The other clip had been placed transesophagoscopically on the mucosal esophagogastric junction. It is seen that the distance between the clips varies considerably from time to time, proving that the two mural layers have migrated over one another.

By roentgenography it was found that the esophagus was uniformly dilated to a small extent, and that there was a shelf-like partial obstruction within the ampulla. The barium suspension passed this obstruction slowly, and after a few minutes the esophagus was empty.

Upon esophagoscopic examination an angry, ulcerated, tumor-like plug was encountered at the level of the diaphragmatic hiatus (Fig. 2). This had

the appearance of a large spherical polypoid tumor. It was biopsied and then an attempt was made to pass the esophagoscope beyond the tumor in order to study the extent of involvement. In so doing, the operator pushed the tumor easily ahead of the instrument until a lumen suddenly appeared in its center, and the mass disappeared into the circumference of the gastric wall. It was evident that the tumor was composed of a sleeve of prolapsed gastric mucosa. When the instrument was withdrawn upward into the proximal esophagus and then reinserted into the stomach, all appearances as far as the esophageal mucosa and position of the organs were normal. When the patient was instructed to "bear down", the sleeve quickly squeezed up into the ampulla again, whereupon it could easily be pushed back again into the stomach. A second biopsy specimen was taken from the tumorous prolapse. Both specimens were later found to consist of gastric mucosa of the cardia type, with considerable inflammatory exudate and small hemorrhages into the *lamina propria mucosae*.

Esophagoscopic examination was repeated upon three other occasions and gastroscopy was carried out twice. Each time the esophagoscope was inserted into the distal part of the esophagus, the prolapsed sleeve of mucosa was encountered. It is to be noted that esophagoscopy was regularly accompanied by retching and straining. Upon each occasion the prolapse could easily be reduced and made to reappear. The gastroscopic examinations showed a stomach which was normal except for hyperemia and unevenness of the crescent of the cardia.

Throughout the three weeks of hospitalization, no episode was observed which appeared to indicate acute or persistent subacute obstruction at the cardia. Repeated fluoroscopy, like esophagoscopy, showed the easily reducible nature of the obstruction. In view of the help previously afforded by dilatation, the patient was given 10 dilatations with the Einhorn pneumatic dilator. Again subjective relief was good, and the patient was returned to duty.

Case 3:—A 43-year old white soldier was being studied for a proved duodenal ulcer. During the course of roentgenologic study, it was found that there was esophagogastric mucosal prolapse (Fig. 3). Esophagoscopic examination was normal. There were no symptoms which could be ascribed to the lesion.

Comment

The concept of normal independent mobility of the gastric mucosa over the underlying layers has been well examined and discussed by Forrsell[4] and Brooks and colleagues[1]. In order that there be mucosal prolapse, there must be in addition to mucosal mobility a point of firm attachment between mucosa and underlying layers distal to the prolapsing segment. Otherwise, the mechanisms which encourage prolapse would lead merely to general slipping of the mucosa, still in normal stratal relationships.

There is widespread belief that normally the mucosa of the cardia is tightly bound down by fibrous tissue. This feature is frequently cited in discussions of

the behavior of variceal connections across the cardia in portal hypertension. The evidence is taken entirely from the histologic picture, for this shows considerable fibrous tissue in the submucosa where the mucosa crosses the cardia. The circumstantial evidence furnished by dead anatomy has been freely translated to apply to the physiologic behavior of the living mucosa. But it has been shown[2] that the mucosa of the normal esophagogastric junction may migrate back and forth for a distance of two or three centimeters in a rhythmic fashion. Roentgenologically, it seemed that the excursions involve the mucosal layer only, but this could not at the time be proved.

Subsequently, it has been possible to investigate the matter in one patient. This soldier had a transthoracic exploration of the cardia and repair of a small hiatus hernia. At the time, the surgeon sutured a silver brain-clip to the *muscularis propria* at the left lateral aspect of the junction of esophagus and stomach, as the junction was identified from external appearances. Later the mucosal junction was identified esophagoscopically, a biopsy was taken of it, and a brain clip attached to the biopsy site. The biopsy specimen was found to include the epithelial junction. The patient was then studied roentgenologically, and it was found that the extrinsic clip remained stationary but that the longitudinal distance between the two clips varied considerably from time to time, indicating movement of the layers over each other at the cardia (Fig. 4). The maximum excursion was two centimeters. There was, then, automatic or spontaneous propulsion of the mucosal layer at the cardia in this patient.

It is believed that the results of this and previous clip studies, by illustrating mucosal mobility in the region, give some insight into the rarity of mucosal prolapse at the cardia. To reiterate, development of prolapse appears to presuppose a point of mucosal fixation distal to the segment of potential prolapse.

As to the propulsive mechanism responsible for retrograde mucosal movement, it is noted that reverse gastric peristalsis has been proposed[3], but it seems doubtful if reverse peristalsis can even rarely be of any propulsive consequence in the fundus and cardia of the stomach. It seemed clear enough from the esophagoscopic observations in the present cases that merely altering the thoracic-abdominal pressure gradient through the Valsalva maneuver is sufficient when the anatomy will permit prolapse.

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THE FEMALE PATIENT WITH DUODENAL ULCER*

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Introduction

Duodenal ulcer is the most frequent cause of chronic indigestion resulting from organic disease in the adult male patient. The purpose of this study is to determine how frequently does duodenal ulcer occur in the female patient. Are there special problems in diagnosis? What effect does pregnancy or the menopause have on the course of the disease? What are the complications and the response to treatment?

Incidence

In an analysis of 1,685 patients with peptic ulcer seen in private practice it was found that there were 1,274 male patients with 411 female patients (Table I).

TABLE I

Patients with Peptic Ulcer

Male	1274
Female	411
	1685

There were 1,505 patients with duodenal ulcer, 1,160 males and 345 females (Table II).

TABLE II

Patients with Duodenal Ulcer

Male	1160
Female	345
	1505

Of a total of 180 gastric ulcer patients there were 114 males and 66 females (Table III).

TABLE III

Patients with Gastric Ulcer

Male	114
Female	66
	180

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Previously it was noted that gastric ulcer occurred with about the same frequency in both sexes but that duodenal ulcer was much more frequent in the male patient.

At the present time the relative preponderance of male duodenal ulcer patients to female duodenal ulcer patients is less.

Hosoi and Alvarez[4] found that in Europe and in America duodenal ulcer occurred three and one-half times as frequently in men as in women.

Craig[3] stated: "Although duodenal ulcer in females has been a steady increasing cause of death the mortality rate from gastric ulcer has fallen fairly steadily and uninterruptedly."

My own figures indicate a steadily rising incidence of duodenal ulcer in the female patient. If only the figures for the last three or four years were used the ratio of male to female duodenal ulcer patients would be less than three to one.

Etiology

Why does there seem to be a greater incidence of duodenal ulcer in the female patient?

Although the general impression seems to be that a peptic ulcer is found only in the high pressure executive male or career woman, actually peptic ulcer occurs in many different types of individuals regardless of sex, occupation or position in life.

Cigarettes, cocktails and "coffee breaks" have been blamed for the apparent increase in peptic ulcer in women.

The "coffee break" has resulted in a higher rate of coffee consumption and cigarette smoking among women workers. Coffee in particular stimulates acid secretion. Coffee and cigarettes are generally taken during a short "coffee break" without food to act as a buffer for the acid.

No statistical correlation has been found between smoking and the incidence of peptic ulcer. Batterman and Ehrenfeld[2] found that smokers with active peptic ulcer on medical management with antacids showed a poor response to treatment and a high incidence of recurrences, as compared to nonsmokers or to smokers who discontinued smoking during the period of treatment. The substitution of cigarettes with low nicotine content resulted in improvement and a lower incidence of recurrences in a group of these patients.

The moderate use of tobacco, alcohol or coffee by adults does not seem to produce ulcer, or to aggravate every ulcer. Smoking, alcohol or coffee may each contribute to delay in healing of peptic ulcer or to its recurrence, but

it would seem that they do not play more than a contributory role in its pathogenesis.

It is generally assumed that chronic excitation of the stomach by emotional tension is the mechanism by which ulcer is produced. The constant hurry and worry and emotional tensions resulting from problems of modern living, the raising of families and maintaining a home in a world threatened by atomic destruction are probably important etiologic factors.

Women no longer live a cloistered existence. They engage in business and industry and are active in club and political work.

Clinical Features

When a female patient consults a physician because of digestive complaints the possibility of gallbladder disease will be given first consideration. The fact that gallbladder disease is more common in the female patient has been so generally recognized that the possibility of other organic upper digestive disease is seldom kept in mind.

Duodenal ulcer is the most common organic cause for indigestion in the adult white male and it is the diagnosis that is first thought of by the physician.

In the female patient duodenal ulcer is seldom considered in the diagnosis.

In the male patient with an uncomplicated duodenal ulcer a typical history is frequently obtained. The symptoms of duodenal ulcer in the female patient are usually not typical. The history is usually indefinite and inconclusive. In general women complain less of their ulcer distress than men. The so-called hunger distress is seldom heard. Sometimes the first inkling of trouble in a female patient is a gross hemorrhage. Nausea is sometimes present and seems to have the same food relationship as does the hunger pain of the male patient. Thus the nausea occurs when the stomach is empty and is relieved by food intake. The nausea is a pain equivalent. In the male patient when nausea is a presenting symptom there is usually gastric retention, and relief of the retention usually relieves the nausea.

Roentgen Study

In the roentgen study the findings depend on the extent of the lesion present, and have nothing to do with the sex of the patient. Ulcer craters both large and small—shallow and deep have been demonstrated. More deformed duodenal bulbs with fewer symptoms occur in the female patient than in the male.

In some female patients the first recognition of the presence of duodenal ulcer is the roentgen demonstration of an ulcer. A history then obtained in retrospect may reveal suggestive complaints.

Complications

The female patient with duodenal ulcer has the same complications as the male, namely hemorrhage, obstruction and perforation.

In 345 female patients with duodenal ulcer there were 23 patients with hemorrhage, 21 with obstruction and 1 with perforation (Table IV).

TABLE IV

Female Duodenal Ulcer Patients with Complications

Hemorrhage	23
Obstruction	21
Perforation	1
	45

In the male group of 1,160 patients with duodenal ulcer there were 72 patients with hemorrhage, 43 with obstruction and 12 with perforation (Table V).

TABLE V

Male Duodenal Ulcer Patients with Complications

Hemorrhage	72
Obstruction	43
Perforation	12
	127

These figures indicate that hemorrhage occurs as a complication about equally in both sexes.

Obstruction is a more frequent complication in the female patient. Perforation is more frequent in the male patient, in this study about 12 times as frequent. Although obstruction was noted more frequently in the female patient only two of these patients were treated surgically. The medical treatment of obstruction in the others relieved the condition. In the male group 12 patients had to be treated surgically for obstruction because the obstruction persisted in spite of medical therapy.

There were 22 patients who had both gallstones and duodenal ulcer. There were 11 male patients and 11 female. Thus, this possibility must be kept in mind and the greater incidence in the female patient recognized (Table VI).

TABLE VI

Duodenal Ulcer Patients with Gallstones

Male	11
Female	11
	22

Duodenal Ulcer in Pregnancy

It has been widely accepted that pregnancy exerts a beneficial effect on peptic ulcer.

Sandweiss[9] reported that in 70,310 consecutive admissions of pregnant women to five Detroit hospitals in a ten-year period, only one proven case of active peptic ulcer in pregnancy was recorded.

Sandweiss[9] reviewed a series of 46 women with proven peptic ulcer, 25 of whom had one or more pregnancies. These 25 women were pregnant 52 times. Only one had active ulcer symptoms during one of her two pregnancies. This woman gave birth prematurely to a living child. With the onset of her second pregnancy, she became symptom-free and carried to full term. Eleven of the 25 female patients stated that their ulcer symptoms disappeared with the onset of pregnancy.

Sandweiss[10] reviewed the medical literature up to 1943 and found only 13 maternal deaths from hemorrhage or perforation of peptic ulcer during pregnancy at term or in the puerperium with the diagnosis confirmed at autopsy. Sandweiss[10] added the 14th case. Anderson[1] added the 15th case, a patient with a perforated duodenal ulcer. Johnston[5] added two more patients, one with a perforated duodenal ulcer and one with hemorrhage. Of these 17 patients reported, 10 had duodenal ulcer and 7 gastric ulcer. Twelve died from perforation and 5 from hemorrhage. Thus, the possibility of these serious complications during pregnancy at term or in the puerperium must be kept in mind.

Rider[8] emphasized that exacerbations of peptic ulcer during pregnancy seem more frequent in the third trimester and not infrequently are complicated by hemorrhage or perforation. The tendency for ulcer distress to subside early in the pregnancy and for symptoms to occur late in gestation seems to be correlated with variations in gastric secretion. Thus, many observers report decreased output of hydrochloric acid during the first six months of pregnancy and increased gastric acidity during the third trimester and in the puerperium.

Opsahl and Long[7] found that significant quantities of ACTH are produced by the placenta. The discovery of placental ACTH and the known effect of ACTH on peptic ulcer tend to discredit the concept of the beneficial effect of pregnancy on peptic ulcer and might explain the occasional duodenal ulcer perforation or hemorrhage which has been reported, perhaps as the result of overproduction of ACTH by the placenta at term.

Menopause and Duodenal Ulcer

Kirsner[6] observed that peptic ulcer in women often develops or recurs with the onset of natural or artificial menopause. The epigastric distress may occur as a direct result of the menopause, or the digestive disturbances may be

primarily the result of peptic ulcer. The possible changes in gastric secretion accompanying the menopause have not been investigated. Such studies might be of considerable interest in relation to the effect of sex hormones upon the output of acid. Hyperactivity of the anterior lobe of the pituitary gland, deficiency of estrogens and other hormonal imbalances may be implicated in the "menopausal ulcers" of women. In evaluating these factors, however, the important role of the emotional disturbances accompanying the menopause should not be underestimated. These emotional disturbances may very well be the factor in the aggravation of ulcer symptoms during the menopause.

Treatment

The medical treatment of the female patient with duodenal ulcer is the same as that of the male patient. The response is usually more rapid and more lasting. The patient is frequently in a better position to follow an ulcer program.

The following case records are briefly presented:

Case 1:—A white female, age 44, seen in November 1944, complained of abdominal pain which had been present for three years. The pain wakened the patient at night. Cabbage, sauerkraut and onions caused distress. The patient was afraid to eat. Complete studies of the digestive tract revealed an active duodenal ulcer. The gallbladder and colon studies were normal.

The response to a medical regime with antispasmodics and antacids was only fair. Recurrences were noted periodically on treatment. In December 1946, a severe hemorrhage occurred. The patient was hospitalized and treated with repeated blood transfusions, diet and antacid medication.

A second hemorrhage occurred three months later, in February 1947. Roentgen studies after the second hemorrhage revealed evidence of obstruction at the pylorus. In May 1947, a posterior gastroenterostomy and a transabdominal bilateral vagotomy was done. Her response to surgery was good.

One year later in June 1948, the patient returned with symptoms of pain which was relieved by strict ulcer diet and medication.

In June 1949, another recurrence was treated with diet and medication.

In June 1951, roentgen studies revealed a nonfunctioning gastroenterostomy and a gastric ulcer was present on the lesser curvature. A medical program for the gastric ulcer was unsuccessful. A subtotal gastric resection was done on October 12th, 1951. When last seen the patient was doing well.

Case 2:—A white female, age 46, seen August 1953, complaining of gaseous distress, vomiting and pain. Pain was present in the midepigastrium and back. The patient was very constipated. The roentgen studies revealed an active duodenal ulcer with a crater and evidence of obstruction. The gallbladder

studies and colon studies were normal. Ulcer diet, tincture of belladonna and antacid were given. The response to therapy was poor.

One month later the patient noted tarry evacuations. The patient was hospitalized. Anemia was present. Ulcer diet and blood transfusions were given. Obstruction persisted as noted on roentgen examination.

The patient was treated for another two months medically. The response to treatment was poor. In December 1953, a subtotal gastrectomy was done. The result to date has been satisfactory.

Case 3:—Patient, a white female, age 63, was seen January 21st, 1954. Patient gave a long history of stomach trouble, diagnosed as duodenal ulcer. In January 1952, roentgen studies revealed a chronic duodenal ulcer with no apparent activity.

In January 1953, because of severe asthmatic bronchitis the patient was given Cortone mg. 25 q.i.d. This therapy was continued from January to May but with gradual reduction in dosage. There was no stomach distress at this time.

In November 1953, while in Florida the patient had a recurrent attack of asthma and Cortone was reordered. At this time her stomach complaints recurred.

The present complaints continued and the roentgen studies revealed the presence of an active duodenal ulcer with a large crater. The failure of response to a careful medical regime finally necessitated a subtotal resection. The patient's response to surgery has been good.

Case 4:—Patient was a white female, age 32, first seen in January 1939, complaining of gas pains. Food relief, and recurrences in the spring and fall had been noted by the patient. Milk helped while other foods such as fatty and fried foods disagreed. Complete studies revealed a normal gallbladder and an active duodenal ulcer.

An ulcer diet with an antacid powder relieved the distress.

In 1943, nine years later a slight recurrence was easily controlled by diet and medication.

In 1948, distress wakened patient at night. Gastrointestinal roentgen studies revealed a recurrence of the duodenal ulcer. The ulcer symptoms responded promptly to medical treatment. The patient has remained well.

Case 6:—Patient was a white female, age 20, seen for the first time in 1939 complaining of epigastric distress usually two to four hours after eating. Her father and brother had duodenal ulcers. A diagnosis of duodenal ulcer was suggested after examination and confirmed by roentgen studies. An ulcer diet and medication gave prompt relief. The patient had recurrent attacks of ulcer

activity in 1940, 1944, 1950, and 1952, easily controlled by diet and medication. At present the patient is doing well on an ulcer regime.

Case 7:—Patient was a white female, age 40, seen July 14th, 1954, complaining of severe nausea and considerable gas and pain in the epigastrium. The roentgen studies revealed an active duodenal ulcer. The response to a medical regime was excellent. The nausea, pain and bloating disappeared completely and promptly.

Case 8:—Patient was a white female, age 42, seen in June 1952. The patient complained of eight years of stomach trouble. The distress occurred 2-3 hours after eating. Vomiting occurred occasionally. The roentgen studies revealed an active duodenal ulcer with crater formation. An ulcer diet with tincture belladonna and an antacid powder gave prompt relief. The patient felt good on this program for 1½ years, then had a mild recurrence after a dietary indiscretion which responded quickly to medical treatment.

Case 9:—Patient was a white female, age 8½ years, seen in April 1954, complaining of distress in the abdomen which was usually relieved by food intake. The attacks of pain were quite severe. Daily distress was present usually several hours after eating. The roentgen studies revealed a duodnal ulcer. The response to medical regime was very good.

Summary

1. Duodenal ulcer occurs with sufficient frequency in the adult female to merit attention.

2. In the female patient with indigestion a complete gastrointestinal roentgen study is essential in establishing the correct diagnosis.

3. The response to medical treatment of the uncomplicated duodenal ulcer in the female patient is good.

4. Hemorrhage and obstruction are important complications, and perforation is a rare complication in the female patient.

5. Pregnancy usually has a beneficial effect on the course of the disease but perforation and hemorrhage of duodenal ulcer at term have been described.

6. Duodenal ulcer may develop or a previously existing ulcer may become reactivated during the menopause.

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Discussion

Dr. Louis T. Kirchenbaum (New York, N. Y.):—What physiological interpretation do you place upon the symptom of nausea as a substitution for a symptom of pain, in that it fails to be relieved by the anticholinergic drug but is relieved by thorazine. The anticholinerigic drug relieves pain but not the nausea.

Dr. Shaiken:—I cannot give you the physiological explanation. My only reason for bringing up the subject of nausea at all was that I have been impressed that in some female patients nausea seemed to be the dominant complaint, and the nausea was very severe and intractable, but the very fact that it was related to the food intake suggested that it might be a pain equivalent. When the patient volunteered the information that the taking of a small amount of food generally helped the nausea, it suggested its relationship to the hunger pain sequence.

In these very same patients pain was not a feature, and my only reason for bringing it to attention at all was that the same thing has not been evident in the male patient. If the male patient does complain of nausea, usually I have been able to find some evidence of gastric retention. In this particular patient I cannot explain why the thorazine relieved the nausea and the anticholinergic drugs did not relieve it.

OPERATIVE CHOLANGIOGRAPHY*

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Operative cholangiography is the visualization of the intra- and extrahepatic ducts by means of a radio-opaque medium for the purpose of demonstrating pathology in the bile-drainage system.

The common duct is hidden in the gastrohepatic ligament and buried against the posterior abdominal wall, and is overlaid by the adjacent viscera. The distal end curves behind the duodenum and passes by the side of the head of the pancreas, often penetrating the head of the pancreas, and then buries itself in the posterior wall of the duodenum for the last part of its course before opening through the ampulla of Vater. As a result of this location and course, the distal end is extremely difficult to visualize as well as to palpate. In some obese patients even the best of surgeons cannot be too sure of what his hands are feeling, since his eyes never see the entire duct. Therefore, exploration of the intra- and extrahepatic biliary passages by visualization and palpation is inaccurate and often inadequate. Most surgeons will accept the passage of a probe or dilator through the ampulla as conclusive evidence of the absence of stones in the distal end of the common duct. Often the exploration of the proximal ducts is ignored. We have shown frequently that it is possible to pass a probe into the duodenum in the presence of stones located in the distal end of the common duct.

Conversely, the inability to pass a probe into the duodenum does not always indicate pathology. There are many variations in size, length, thickness, course, direction, and curvature of the biliary ducts. Anomalies may also be present. There may be a physiologic spasm of the sphincter of Oddi. In any difficult case, true appraisal of the anatomy of that particular patient is almost impossible.

In some cases it is impossible to tell with accuracy whether the probe has or has not passed into the duodenum. The surgeon may be misled into thinking that the probe has passed into the duodenum, when actually the posterior wall

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of the duodenum has merely been invaginated into the lumen. We have demonstrated this by opening the duodenum over a probe which we thought had passed the sphincter only to find the posterior wall had been invaginated into the lumen of the duodenum.

Gallstones are harbored by approximately one out of ten adults. Stones can be present in the common bile duct without causing pain or jaundice. Crump[1] has shown from autopsy findings that approximately 25 per cent of adults who have cholelithiasis will have stones in the biliary ducts. These will vary in size

Fig. 1—Cholangiogram showing normal duct. The duct is not dilated and comes to a fine point. No dye has entered the duodenum due to a spasm of the sphincter of Oddi.

and number from the solitary, large stones to the multiple small stones which may extend proximally in the hepatic ducts into the liver itself.

The major complications of biliary surgery are injuries to the bile ducts and stones that are left within the ducts. Stones are left in about 10-18 per cent of the cases because the duct was explored inadequately or not at all[1,3,4].

In an analysis of 350 cases of direct exploration of the common duct at Touro Infirmary, New Orleans, La., in 10 per cent of the cases stones were overlooked.

Except for stressing adequate exposure of all structures in the area and deliberate careful technic, there has been no major contribution to the prevention of complications of biliary surgery in recent years except operative cholangiography.

Indications for Common Duct Exploration

Our indications for exploring the common duct are more liberal than those held generally. We have divided them into two groups: the definite and the indefinite.

Definite:—The definite indications include:

1. All cases of obstructive jaundice.
2. Palpable stones in the common duct at the time of surgery.
3. Dilated common bile duct found at the time of operation.
4. Multiple small stones in the gallbladder with an enlarged cystic duct.

Indefinite indications:—The indefinite indications may be listed as:

1. A thickened common duct.
2. Questionably enlarged common duct.
3. Thickening of the head of the pancreas.
4. Evidence of chronic pancreatitis.
5. A thick-walled contracted gallbladder.

In the presence of indefinite indications, cholangiography should be performed through the cystic duct to determine whether or not the common duct should be opened.

Operative cholangiography should follow *every* surgical exploration of the common duct, regardless of the findings, since it is an added means to determine pathology within and patency of the bile duct. It is an adjunct to but not a method of replacement for surgical exploration when the indications for exploration are definite. When the indications are indefinite, cholangiography may prevent a needless surgical exploration of the common duct.

History

Mirriz[5] was the first to employ operative cholangiography. He presented a summary of the method in 1932. It was adopted and strongly advocated by Hicken and Best[6-8] in this country and has occasioned many articles subsequently. Many others have advocated its routine use. Despite many enthusiastic reports, the method has not received the widespread adoption it merits.

There are a number of reasons for hesitancy in utilizing this aid during surgery.

a. Many smaller hospitals still lack portable x-ray equipment.

b. Some surgeons are prone to speed rather than to thoroughness, and hesitate to prolong any operative procedure.

c. Others have the misconception that the method cannot offer much more than the passage of a probe, direct visualization, and palpation in evaluating the pathology present.

Fig. 2

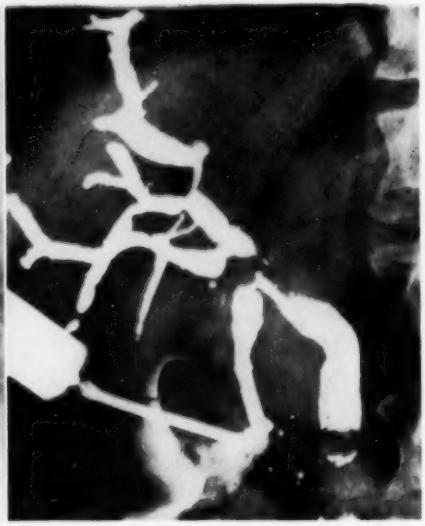

Fig. 3

Fig. 2—Cholangiogram showing dilatation of the common duct due to an obstruction.
Fig. 3—Cholangiogram showing blunting at end of common duct caused by a large stone.

d. Until recently the usual radio-opaque medium used was 35 to 70 per cent iodine in oil. This solution is prone to give false negative shadows and completely obscure small stones.

Media

Most of the available media for cholangiography are unsatisfactory. This may be due to the high concentration of iodine which casts too heavy a shadow, or because of a thick oily base making the solution too viscid and immiscible with bile, or a watery base making solution too thin. An ideal medium should have the following characteristics:

1. It should cast a shadow of such intensity that enables visualization of all stones present. Highly concentrated iodine solutions cast heavy shadows which will obscure small stones if the solution surrounds them completely.

2. It should be nontoxic and nonirritating to the patient.

3. It should be freely miscible with bile to allow thorough mixing.

4. Surface tension should be low enough to prevent the trapping of air, since this will give the false shadows mentioned previously.

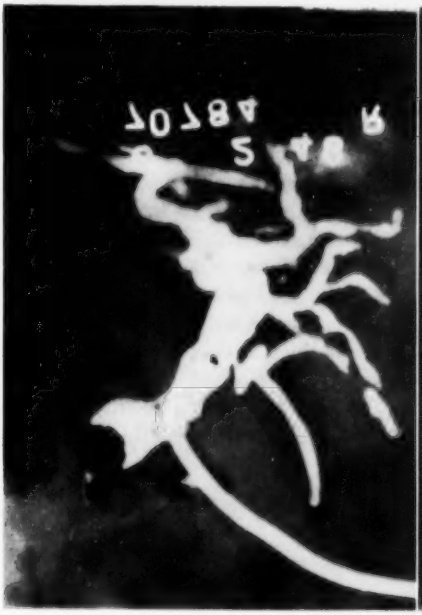

Fig. 4

Fig. 5

Fig. 4—Cholangiogram showing a crescentic defect of the distal end of the common duct caused by a large stone.

Fig. 5—Cholangiogram showing moderate dilatation of common duct and a negative shadow which proved to be a stone.

5. It should have proper viscosity, that is, low enough to be injected through a small-bore needle (22-24) and high enough to be retained within the common and hepatic ducts.

None of the media now available for cholangiography fulfills all these requirements. If such a medium were available, many of the objections to operative cholangiography would be overcome. In an endeavor to find a more satisfactory solution we have developed a medium which more nearly fulfills the ideal requirements than any solution now available. Space does not permit

the detailed description of our experimental work. This work may be summarized by saying that a solution containing 39 per cent Neo-Iopax (Schering) and 4 per cent Dextran was the best suited for this purpose.

Technic

The technic of operative cholangiography is extremely simple, requires no special equipment other than a portable x-ray machine and a cassette holder, and adds only 15 minutes to the operating time. Our patients are routinely given atropine gr. 1/150 and seconal gr. 1½ preoperatively. They are not given morphine, Demerol, or related opiates which on occasion cause spasm of the sphincter of Oddi. A cassette holder is placed under the patient so that the upper edge is even with the nipple line. Following the incision the skin towels are sutured to the skin edges so that the metallic instruments will not cast obscuring shadows on the x-ray plate. The abdomen is entered through the incision preferred by the surgeon and is explored. If the gallbladder is to be removed, a clamp is placed across the fundus and it is pulled up into the wound. The cystic artery, cystic duct, and common duct are identified and exposed. The cystic artery is then divided between clamps and ligated, allowing straightening of the cystic duct. At this time it is determined whether any of the definite or indefinite indications for exploration of the common bile duct are present. If definite indications are present, the common duct is opened longitudinally and explored in the routine manner, and any stones present are removed. In any event, whether stones are found or not, the common duct is irrigated and a "T"-tube is inserted. A cholangiogram is then made through this "T"-tube. While the x-ray plates are being processed, the gallbladder is removed.

When indefinite indications exist, a preliminary cholangiogram is made through the cystic duct. Stones are less likely to be found when the indications are indefinite, and cholangiograms made through the cystic duct have definite advantages:

1. It may shorten the procedure by making a formal exploration of the common duct unnecessary.

2. It reduces the number of postoperative hospital days for those patients who would otherwise have had common duct drainage.

3. Trauma and the likelihood of a stricture of the common duct is reduced.

Our technic for cholangiography by either the "T"-tube or cystic duct method is as follows: The amount of dye to be injected is first estimated. This is governed mainly by the size of the common duct. We have found that the average biliary system, exclusive of the gallbladder, will hold from 10 to 15 c.c. With moderate dilatation it will hold up to 30 c.c., and with marked dilatation it will hold from 40 to 50 c.c.

If the cholangiogram is to be made after exploration of the common duct, the dye is injected directly through the "T"-tube. If the cholangiogram is to be made through the cystic duct before exploration, a small transverse incision is made in the upper ⅓ of the cystic duct. Several sizes of ureteral catheters are sterilized and one, which will pass readily, is threaded through the cystic duct into the common duct for a distance of one to two cm. A ligature is passed around the cystic duct below the incision and the catheter is tied in place so that the system is water tight. Leakage at the time of injection will make interpretation difficult.

Two injections are used routinely. Regardless of the size of the duct, the first injection is always 5 c.c. Small stones may be obscured by large amounts of the dye, especially if the dye completely surrounds the stones. An x-ray plate is made after this injection. In rapid succession second and third plates are made following the additional instillation of 15 to 50 c.c. of the dye, depending on the amount of dilatation of the ducts. All metallic clamps and instruments must be removed from the field while the x-rays are being taken. If the cystic duct is used, the catheter is removed after the third plate and the duct is ligated. The gallbladder is then removed while the plates are being processed. We do not recommend the routine injection of dye directly into the common duct with even the finest of needles for three reasons:

1. The hole made by the smallest hypodermic needle will permit the leakage of the dye into the free peritoneal cavity, obscuring the duct.

2. Bile can leak into the free peritoneal cavity.

3. The needle may penetrate the posterior wall of the common duct and enter the portal vein. Occasionally we do use this method, and have never had a serious complication resulting from it.

Interpretation of Films

With experience the surgeon can become proficient in interpreting his own cholangiograms. A normal common duct is not dilated, and comes to a fine point at the ampulla of Vater (Fig. 1). A duct containing stones may show the following:

1. The duct may be dilated (Fig. 2). Dilatation may exist from other causes, but when present one should be highly suspicious of the presence of stones.

2. Blunting of the distal end (Fig. 3).

3. Cresenteric defect of the distal end of the common duct (Fig. 4).

4. Negative shadows anywhere within the bile ducts (Fig. 5). The dye should fill the intrahepatic bile ducts to rule out any intrahepatic duct stones.

5. When the dye does not enter the duodenum, special consideration is necessary. In the absence of mechanical obstruction or physiologic spasm of the sphincter of Oddi, the dye should pass readily from the common bile duct into the duodenum. If the end of the common duct comes to a smooth point and no dye enters the duodenum we assume that the obstruction is caused by a physiologic spasm, and that no intrinsic pathology is blocking the duct.

Comment

By the use of cholangiography we have found stones in about 4 per cent of cases in which they would otherwise have been overlooked.

It is difficult for us to understand many of the objections raised against operative cholangiography, since the majority of stones left in the common duct after exploration are admittedly found on the routine cholangiogram taken usually on the 10th postoperative day. No experienced surgeon would dare remove a "T"-tube without a cholangiogram. It seems most logical to us, if cholangiography is done at all it should be done at the time of surgery. Often there are multiple stones in the common duct, occasionally numbering in the hundreds, especially when the intrahepatic ducts are filled. In such cases it is very easy to overlook one or more stones. The serious complication of leaving a stone within the common duct often leads to a second operation which unquestionably raises the mortality and morbidity of biliary surgery.

Summary

1. Operative cholangiography will reduce the number of stones left in the common duct.

2. The definite and indefinite indications for exploration of the common bile duct have been listed.

3. When the definite indications exist, the cholangiogram is made through the "T"-tube following exploration.

4. In the presence of indefinite indications, a preliminary cholangiogram is made through the cystic duct. Should the cholangiogram reveal the presence of stones, they are removed and a repeat cholangiogram is taken.

5. The technic of cholangiography has been described.

6. Many of the objections to operative cholangiography have been obviated by the development of a water soluble, slightly viscid solution.

7. We feel that this procedure will reduce the operative mortality and morbidity in biliary surgery by reducing the number of reoperations.

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DISCUSSION

Dr. I. R. Jankelson (Boston, Mass.):—I should like to ask Dr. Kaplan a question. In how many cases of cholangiography does the pancreatic duct fill and what is its significance?

Dr. Arthur A. Kirchner (Los Angeles, Calif.):—How many times does cholangiography fill the pancreatic duct and, when it does, what is the significance?

Dr. Kaplan:—In performing operative cholangiography it often does fill the pancreatic duct. It enters it in about 30 per cent of the cases. It has no significance. We have never seen acute pancreatitis follow or occur in an individual in which the pancreatic duct filled.

Dr. James T. Nix (New Orleans, La.):—I should like to ask Dr. Kaplan, first, was there any increase in mortality due to using the operative cholangiogram and, second, were there any allergic manifestations to the use of the dye?

Dr. Kaplan:—It has not increased the mortality, and we have done about 250 cases. There was one case that developed jaundice following a cholangiogram, but in going back over the case, we were not sure whether the patient was developing jaundice because she was obstructed and then had subsided, or whether this was just an exacerbation of a latent jaundice. In our series of cases there have been no allergic phenomena.

Dr. Joseph Shaiken (Milwaukee, Wisc.):—May I ask Dr. Kaplan what has been his experience with the use of cholografin in these cases?

Dr. Kaplan:—I have had no experience with it.

Dr. Nathan Brodie (Brooklyn, N. Y.):—What has been the incidence of error in operative cholangiography at the time of surgery? Further, was this incidence of error established by palpation? In regard to the figures quoted,

of 10-15 per cent, I am sure that many of the clinics, and most surgeons, run a lower incidence of undisclosed common duct calculi down to perhaps 5 per cent. Again, my question is in regard to your incidence of error.

Dr. Kaplan:—The incidence of error in our series of cases runs around 4 per cent. This includes cases which we call "trapped air", and instances in which the cholangiogram was not diagnostic.

In the cases in which the cholangiogram was not diagnostic and the possibility of a stone in the common duct existed, the duodenum was opened and the duct explored retrograde. Following this technic, we have left no stones behind.

GASTROSCOPIC OBSERVATIONS OF THE ANTICHOLINERGIC EFFECT OF PRO-BANTHINE® ON GASTRIC MOTILITY AND PYLORIC FUNCTION*

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New York, N. Y.

The recent literature has been replete with the evaluation of anticholenergic drugs. Most of these investigations were limited to their effect on the stomach. The majority of these concerned themselves with secretory studies and relatively few have been confined to gastric motility and pyloric function. Clinically, a great deal of significance has been attributed to gastric hypermotility and pylorospasm.

Recently, some objective studies have been carried out on gastric motility. These, in a measure substantiated some clinical impressions. Stempien et al[1] have observed endoscopically that the normal gastric mucosa varies in function from hypo- to hyperfunction. In gastric disease[2] such as gastritis and peptic ulcer, however, increased gastric activity is uniformly present. Hywater et al[3], using the balloon photokymographic technic, have actually correlated antral gastric hypermotility with epigastric pain. They also demonstrated that Banthine inhibited hypermotility and allayed pain. Similar findings were reported by Abbot et al[4]. They made similar observations on the gastric fistulous subject "Tom", after introduction of Banthine 50 mg. Another contribution on the effect of anticholenergic drugs on gastric motility was made by Hawkins et al[5]. Using the x-ray technic, they demonstrated "marked delay in gastric emptying".

With these studies in mind, this investigation was undertaken with a two-fold purpose. First, to substantiate previous findings. Second, to amplify our knowledge on this subject. Some of the questions we sought to answer more definitely were: What degree of gastric inactivity is produced by the recommended doses? What dose is required to produce relative complete inactivity? How soon after administration is the drug effective? What is the difference between the parenteral and oral administrations? For how long is gastric inactivity maintained from a single administration of the drug? Finally, how can we benefit in treating our patients from this added knowledge?

The gastroscopic method of investigation was selected because it was felt that it was objective and would therefore be most reliable to study gastric motility. Normally, peristaltic waves are observed to start at about the distal third of the stomach, being most pronounced at the incisura. These appear as circular contracting folds moving distally towards the pylorus, in rhythmic

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sequence, about every 18 to 20 seconds. As the contraction wave terminated at the pylorus, the pyloric ring closes suddenly, forming a stellar or puckered appearance. It stays closed for a short period, then it opens and appears as a round dark hole of about ½ to 1 cm. in diameter. About the same time, a new peristaltic wave begins to form and the rhythmic process starts all over again. According to Schindler[6], the pylorus is visualized in 80-90 per cent of the cases.

The measure of the efficacy of the drug in this study is based on its ability to cause gastric motor inactivity. This phenomenon occurred when adequate dosage was administered. Peristalsis ceased rather suddenly and at the same time, the rhythmic opening and closing of the pylorus also stopped, leaving the stomach in a relaxed state. Some observers[4] have used hypo- and hyperactivity of peristalsis as a measure of the degree of effectiveness. It was found

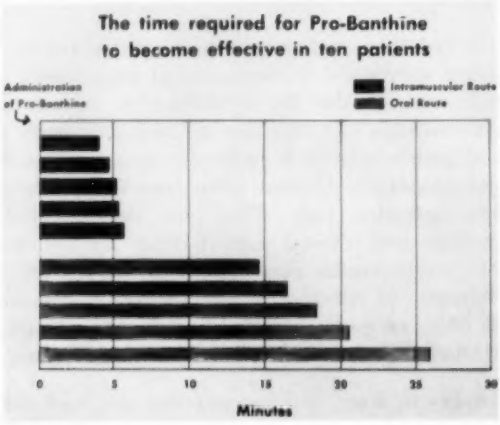

Fig. 1

that such criteria are not reliable, since these variations in gastric activity have been observed in apparently normal individuals[1].

Color changes of the mucosa have also been used as a measure of gastric activity[7]. Such alterations appear as a result of hyperemia and engorgement. Observations of changes in color were also made whenever they were definite.

Selection of Patients

The patients selected for this study consisted of individuals complaining of upper abdominal pain of varying degree. They were all ambulatory and were observed in the out-patient department. A total of 40 patients were studied on whom approximately 50 gastroscopies were performed. This group consisted of ten cases with duodenal ulcer; two with gastric ulcer; five with gallbladder disease; five with chronic gastritis and 18 with functional dyspepsia. Their ages ranged from 17 to 72 years of age. Twenty were males and 20 females.

Method of Study

The patients reported in the morning to the clinic, without breakfast. No pre-examination medication was given, except for 2 per cent Pontocaine, which was used as a surface anesthesia to the hypopharynx. Pro-Banthine was administered intramuscularly to one group and orally to another. In some instances, on different occasions, the same patient was exposed to both types of tests.

Length of Time Required for the Anticholinergic Drug to Become Effective

The first phase of the investigation was undertaken to determine the time taken for the anticholinergic agent to become effective. In one group, the gastroscope was first introduced and the degree of gastric motility observed. The anticholinergic drug was then administered intramuscularly and the time

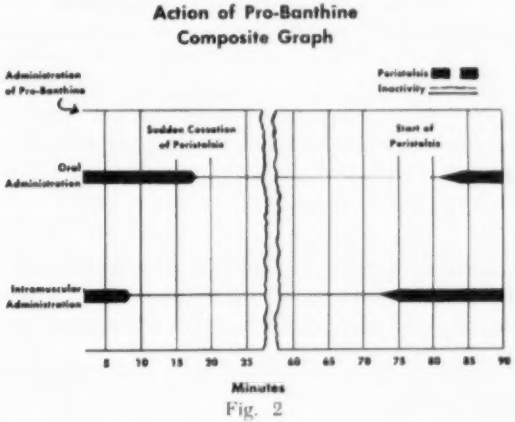

Fig. 2

it required for gastric inactivity to take place was noted. It took approximately 6-8 minutes for this to occur after parenteral administration (Fig. 1).

It was found by trial that it took much longer for the drug to become effective when administered orally. Under the circumstances, to obviate prolonged instrumentation, the medication was given orally and after a 10 to 15 minute wait, the patient was gastroscoped and observations were made. It was found that it took approximately 15-25 minutes for it to become effective by this route (Fig. 1).

Duration of Gastric Inactivity

The second phase of the study concerned itself with the determination of the duration of the effectiveness of the drug. Gastroscopic observations were made on ten cases, to observe the time of return to normal gastric peristalsis.

First, the patient was given an effective dose to cause cessation of gastric peristalsis. After a 45 minute wait, three selected individuals were gastroscoped every 15 minutes, until resumption of peristalsis was observed. It was found that gastric inactivity lasted about one hour. After the approximate duration of inactivity was established, the remaining seven cases had to be instrumented only once. The gastroscope was introduced at the 55 minute period, about five minutes prior to the anticipated resumption of gastric peristalsis and observations were made of the transition from inactivity to activity. This transition was relatively sudden not taking more than one to two minutes. Peristalsis started with a few low amplitude waves 18-20 seconds apart and then followed by normal waves. Out of the 10 cases observed, gastric inactivity lasted 60 minutes in

TABLE I

MINIMAL EFFECTIVE DOSE REQUIRED TO CAUSE GASTRIC INACTIVITY IN 32 PATIENTS

Pro-Banthine Dose in mg.	Route of Administration: Intramuscular	Oral
30	I I	I I I I I I
25	I I	
20	I I	
15	I I I A	A A A A A A A I
10	I I I I	
5	A A A A	

I–Gastric Inactivity
A–Gastric Activity

seven; 65 minutes in two; and 70 minutes in one. The route of administration of the drug did not seem to influence its duration of action (Fig. 2).

MINIMAL EFFECTIVE DOSE REQUIRED TO PRODUCE GASTRIC INACTIVITY

Once the time required for the anticholinergic medication to become effective was established, a study was undertaken to determine the minimal effective dose. This dose is the minimum amount of the drug that would cause cessation of visible gastric peristalsis in an average human subject.

Eighteen patients were investigated using the intramuscular method of administration. We first started with relatively large doses of 30 mg. and then progressively made 5 mg. reductions in the dosage of subsequent cases, until no effect on gastric peristalsis was observed. Gastric inactivity was produced in two individuals receiving the 30 mg. doses; in two with 25 mg.; in two with

20 mg.; in three out of four with 15 mg.; in four out of four with 10 mg.; and in none out of four with 5 mg.

A comparative study was done, evaluating the oral method of administration. Fourteen individuals were observed. Complete gastric inactivity was induced in six receiving a 30 mg. dose. The same result was obtained, however, in only one out of eight patients taking 15 mg. In the remaining seven, no visible change in peristalsis was observed (Table I).

Some color changes of the mucosa were observed in a small percentage of the cases. The change consisted of a varying degree of fading of the normal orange red to pink. This phenomena was seen in only 12 out of 29 cases where gastric inactivity was induced.

Conclusion

Gastroscopic observations show that Pro-Banthine, when administered in adequate doses will cause relatively complete gastric inactivity. Gastric peristalsis stops rather suddenly and leaves the stomach in a relaxed state. At the same time, the rhythmic (pyloric function) opening and closing of the pylorus ceases and a relative paralysis ensues. This ability to cause gastric motor inactivity offers a reliable method for testing anticholinergic drugs in humans. In some instances, the anticholinergic drug will also cause a fading of the orange-red color of the gastric mucosa.

The minimal effective dose varies with the mode of administration. Uniform cessation of peristalsis was obtained with only 10 mg. when given intramuscularly, whereas, as much as 30 mg. had to be used orally to obtain the same effect.

The length of time required for the drug to become effective was also dependent on the mode of administration. It took approximately 6-7 minutes when the medication was given intramuscularly and as much as 15-25 minutes when taken orally. Increased dosage did not seem to affect these time factors.

An important aspect of this study was the determination of the duration of action of the drug. Complete gastric inactivity lasted approximately 1 hour to 1 hour and 10 minutes. For an additional few minutes, diminished peristalsis was observed, then normal gastric activity ensued. Increased doses did not prolong the action of the drug. Both the intramuscular and oral route of administration resulted in approximately the same duration of action.

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Discussion

Dr. Sydney Rothbart (Brooklyn, N. Y.):—I should like to ask Dr. Barowsky, since we know from Wolff's work with his fistula case, about the psychosomatic effects on the stomach, and certainly know even in skilled hands, that the presence of the gastroscope is a foreign body and produces trauma and apprehension, how much percentage of error did you give to the use of this instrument, changes of color, and things of that sort, and trauma. In the presence of a gastroscope some interference with normal function must be produced.

Dr. Barowsky:—It is apparent that the introduction of any foreign body in the stomach, as the gastroscope, may produce some changes, but in this particular study there is no interference with our investigation, for the reason that the introduction of a gastroscope may stimulate peristalsis and not diminish it, and we were concerned primarily with relaxation of the stomach and producing complete gastric inactivity. After doing approximately 7,000 gastroscopic examinations, I cannot recall an instance where actually complete relaxation was obtained in the stomach by the introduction of a gastroscope.

Dr. Rothbart:—I had in mind that you needed twice the dose, 30 mg. to produce this relaxation, which may have been due to the fact that the presence of the gastroscope was producing hyperperistalsis and that is why you had to have twice the advocated dose; that might be one idea.

Dr. Barowsky:—As I stated in my paper, some of my findings were corroborated by Dr. Almy, investigating the sigmoid area, who had to use three times the recommended dose. My cases were in most instances selected patients that I had gastroscoped several times before, and they were willing to come back and have the examinations done again. I don't believe there was a factor of increasing peristalsis in these individuals.

Dr. Cecil Mantell (Staten Island, N. Y.):—I should like to ask Dr. Barowsky if he has repeated his experiment following the use of Belladonna and atropine? If he has, what was the optimal doses of these drugs which he found to be effective?

Dr. Barowsky:—Routinely in doing gastroscopic examinations every patient is given atropine sulfate gr. 1/150 to counteract the vagal reflex. I have administered that routinely to at least five thousand patients. I have never observed

complete gastric inactivity resulting from the above medication. There may be some slowing down of gastric peristalsis, but the only way I was able to obtain complete gastric motor inactivity was to use the anticholinergic drug in adequate doses.

Dr. Heinz Eisenstadt (*Port Arthur, Tex.*):—What dosage of Pro-Banthine would you recommend and how often would you use it?

Dr. Barowsky:—The scope of this paper does not permit me to go into detail concerning the clinical application. We have, since then, applied our findings clinically, and the results have been unusually gratifying.

From our results I would say that one should prescribe at least twice the dose that is recommended by the manufacturers, that is, at least 30 mg. should be used when you want to obtain a physiologic effect.

Dr. Eisenstadt:—How often?

Dr. Barowsky:—You can readily surmize that if the duration of the action of the drug is only one hour, you would necessarily have to use it much more frequently than it is recommended.

Dr. James B. Hammond (*Indianapolis, Ind.*):—I should like to ask Dr. Barowsky if he has extended his studies on the duration of the effect of Pro-Banthine to include doses above the minimal effective dose. I am especially interested to know if there is a longer effect, and I should also like him to comment on what other clinical evidences of parasympathetic blockade were observed clinically in the patients, with the dosages he was using.

Dr. Barowsky:—Increased dosage did not in any way alter the duration of action; in other words, if I gave 30 or 40 mg., the duration of action still was constant. Apparently elimination or destruction of the drug is very rapid and the effect was just the same.

As far as other effects are concerned, I assume you mean side-effects. We have observed very little disturbance resulting from side reactions, except from one type of effect, and that was xerostomia. It was annoying to some of our patients.

INITIAL HEMATEMESIS

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Roslyn Heights, N. Y.

Gastrointestinal bleeding is always a serious situation. So serious is this type of bleeding that both the medical and surgical literature is replete with articles on this subject. The causes for this bleeding may be due to one or several of many different type lesions. The bleeding site may occur anywhere from the esophagus to the rectum. The passage of gastrointestinal blood per rectum is classified as melena or hematochezia and the vomiting of blood

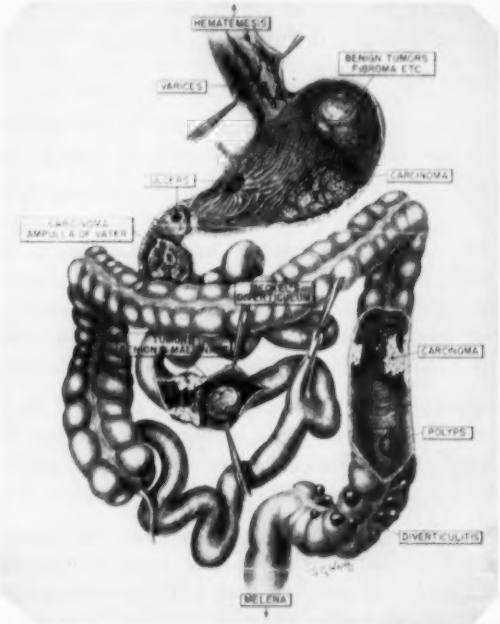

Fig. 1—Drawing illustrating the common causes of gastrointestinal hemorrhage. The expulsion of blood from the mouth is hematemesis and the passage of blood through the rectum is melena. Taken from Chapter 28 by C. B. Ripstein, M.D. in *Emergency Surgery* by B. J. Ficarra, M.D. published by F. A. Davis Co. Philadelphia.

is given the name hematemesis. The accompanying diagram (Fig. 1) presents a pictorial review of the commonest causes for gastrointestinal bleeding.

In this essay thought will be given to hematemesis with special reference to initial hemorrhage and the gravity of the pathology underlying this first gastric

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hemorrhage. Unfortunately, there are very many internists and surgeons who still prefer conservatism in the management of the gastric hemorrhage patient who bleeds for the first time. There are still others who believe that a decision for or against surgery resides within the patient's age group. Surgery has been advised more quickly in the older age group and conservatism is advocated often for the younger adults with an initial gastric hemorrhage.

In the management of patients with initial hematemesis one must judge for himself what the preferred course of therapy should be. Most often a decision for or against operation is based upon personal experience. All surgeons are

Fig. 2

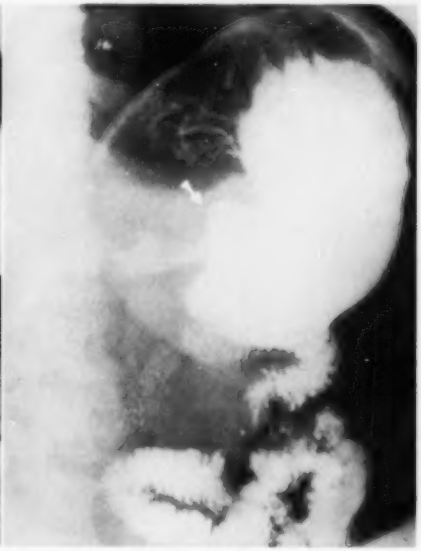

Fig. 3

Fig. 2—Barium gastric study of patient described as Case 1. Gastric ulcer found in lesser curvature. This x-ray was taken in May 1953 after the patient had recovered from her initial gastric hemorrhage.

Fig. 3—Roentgenogram of the stomach of the same patient as figure 2 showing an increase in size of the ulcer. This x-ray was taken three months after the one shown in figure 2.

entitled to their own routine and an "open mind" should be maintained by the nonconformist. This respect for the other fellow's opinion is a characteristic attribute of the American surgeon. So too, in this problem of initial hematemesis some of us have arrived at a definite conclusion and in most cases prefer surgery to conservatism. Few there are who regret operating as soon as possible for initial gastric hemorrhage: but there are many surgeons who regret operating too late.

Early operation for initial gastric hemorrhage is advocated for many reasons. The literature abounds in voluminous articles substantiating these reasons.

Each author gives his reasons and fortifies his statements with case reports and similar data. In this presentation surgery is advocated for a new reason, namely, that the underlying pathology often is more serious than might have been suspected from the history or clinical picture of the patient. Succinctly, it may be stated that initial gastric hemorrhage is usually associated with major underlying pathology and surgical intervention is often the preferred method of identifying and removing the offending pathology. On occasion the failure to remove the cause for an initial gastric hemorrhage may result in subsequent hemorrhagic episodes with the frightening possibility of an associated mortality.

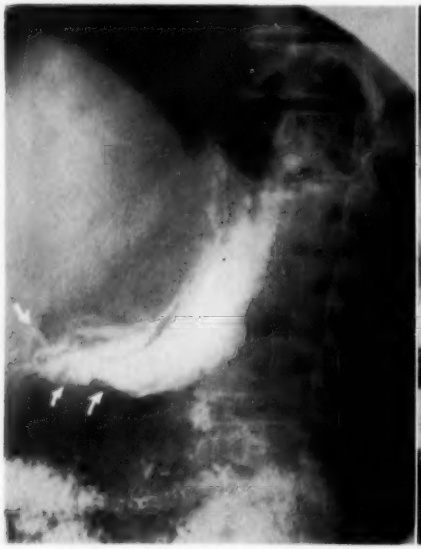

Fig. 4

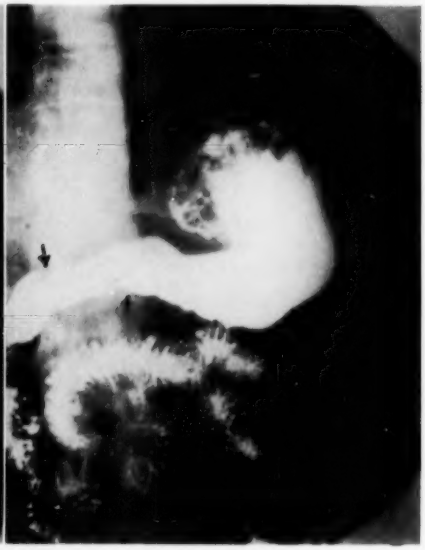

Fig. 5

Fig. 4—Barium x-ray of stomach of patient described as Case 2. Upper arrow indicates a duodenal ulcer. Widening of the mucosal pattern is noted. At operation duodenitis and gastritis plus the ulcer were present.

Fig. 5—Roentgenogram of patient described as Case 3 showing irregular appearance of the gastric antrum. Operation revealed carcinomatous changes in the ulcerated area.

The following case studies illustrate the point in question and evidence is offered in support of the premise that surgical intervention for initial hematemesis of gastric origin is the procedure of choice for most patients deemed operable.

Case Reports

Case 1:—Miss M. C., a 42-year old factory supervisor, had a gastric hemorrhage for the first time in May 1953. She was hospitalized and treated successfully. Part of the treatment consisted of six transfusions of whole blood to

combat the blood loss. Her past history was negative for gastric disease. A barium study of the gastrointestinal tract performed in 1951 during a routine study was negative. After the patient recovered from her initial bleeding a barium x-ray was taken of her stomach and duodenum which revealed a gastric ulcer (Fig. 2).

This patient was advised to have a gastric resection but she preferred to return home in order to enjoy a summer holiday. She did promise, however, that she would return for future surgery. This she did in August 1953. Another

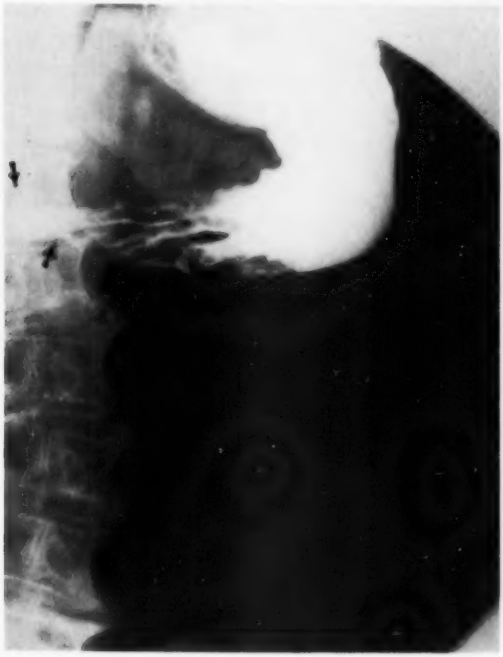

Fig. 6—Barium x-ray of stomach showing antral gastritis, pylorospasm, duodenitis with ulcerations and gastritis in the patient described as Case 4.

gastric series was taken preoperatively which showed a marked progression of the ulcer.

The patient was subjected to an exploratory laparotomy. Gastric carcinoma was found and a total gastrectomy was performed. The patient was discharged from the hospital on a liquid, gruel and jello diet. The diet was gradually increased so that at present she tolerates soft foods. On occasion she has an episode of vomiting following meals which consist of meat. At the time of this writing (February 1954) she has lost 2 lbs. She is not uncomfortable and is ambulatory.

Case 2:—Mr. S. K., a 38-year old postal clerk, was hospitalized for a gastric hemorrhage in June 1953. He was treated conservatively and then discharged from the hospital. Subsequently he was treated medically with diet and banthine —later he was given pamine. In August 1953 he had an exacerbation of pain in spite of diet, drugs and Amphojel. At that time he was seen in surgical consultation for the first time. Barium studies were made of the stomach and duodenum which showed a duodenal ulcer and marked gastritis.

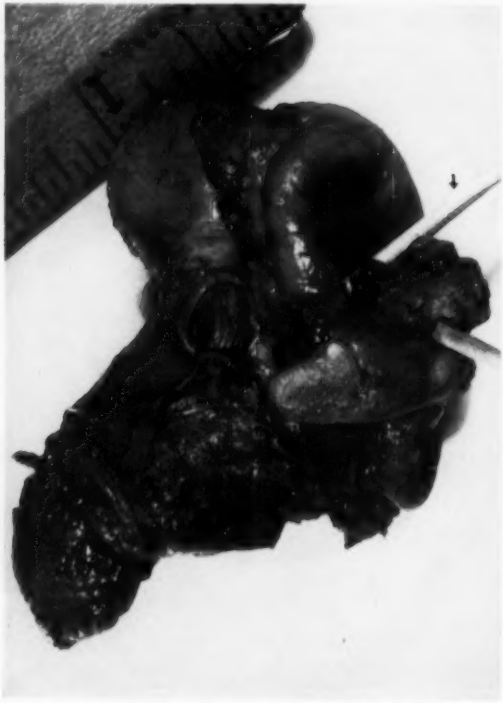

Fig. 7—Operative specimen illustrating a benign duodenal polyp contained in a diverticulum (wooden probe marked by arrow) which caused repeated hemorrhages into gastrointestinal tract plus hematemesis. Patient was a 46-year old female who had a previous gastrectomy for an initial hematemesis. Bleeding continued after her operation. Postoperative x-rays of stomach failed to disclose any lesion. Duodenum opened at second operation and findings were as illustrated.

The patient was advised to have surgery and was hospitalized. A gastric analysis was performed which revealed a marked hyperchlorhydria. The patient was prepared for and subjected to a subtotal gastrectomy plus vagotomy. The operative diagnosis was duodenal ulcer, duodenitis and gastritis. This patient had an uneventful recovery and was discharged two weeks after surgery. He is well and has returned to his former occupation.

Case 3:—Mr. B. B., a 43-year old male store clerk, was seen for the first time in August 1953. His complaints were of several weeks' duration. These were epigastric pain and hematemesis. The hematemesis was not severe. His family physician kept him at home on a diet, antispasmodics and bed rest. Two weeks after his bleeding episode the patient was subjected to complete studies of his gastrointestinal tract. These films revealed antral ulcerations and 20 per cent gastric retention.

Surgery was performed after a preoperative regimen. Diffuse multiple ulcerations in the antral region were found. A frozen section revealed carcinoma-

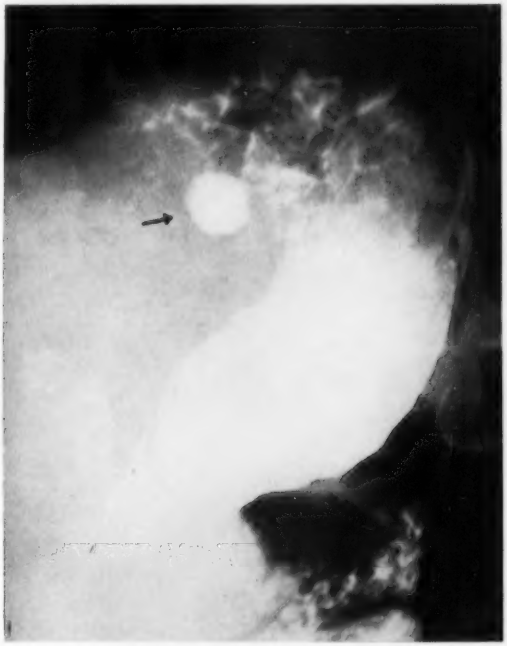

Fig. 8—Roentgenogram of a pseudodiverticulum of the stomach in a 50-year old man with a perforating gastric ulcer found at operation. Patient had a previous history of hematemesis. This type of x-ray picture resulting from a penetrating ulcer has been called Haudek's niche.

tous changes in the ulcerated areas. The regional lymph nodes were negative. The pathologist did not see any invasion. He believed the carcinomatous process to be an early one and sufficiently well localized to allow a high two-thirds subtotal gastric resection. This was performed and the patient had an uneventful recovery.

Case 4:—Mrs. M. M., a 60-year old housewife, had a long history of epigastric distress, intolerance to coarse and spicy foods. She medicated herself

with home remedies and did not consult a doctor until she had a gastric hemorrhage. She was treated at home by her family physician because she refused hospitalization. She recovered from this initial hematemesis after receiving two blood transfusions at home. Two months later (September 1953) she was seen as an office patient. Her complaint at that time was persistent epigastric pain. A barium swallow was given which revealed antral gastritis, pylorospasm and duodenitis with ulcerations. This patient has refused any surgical therapy.

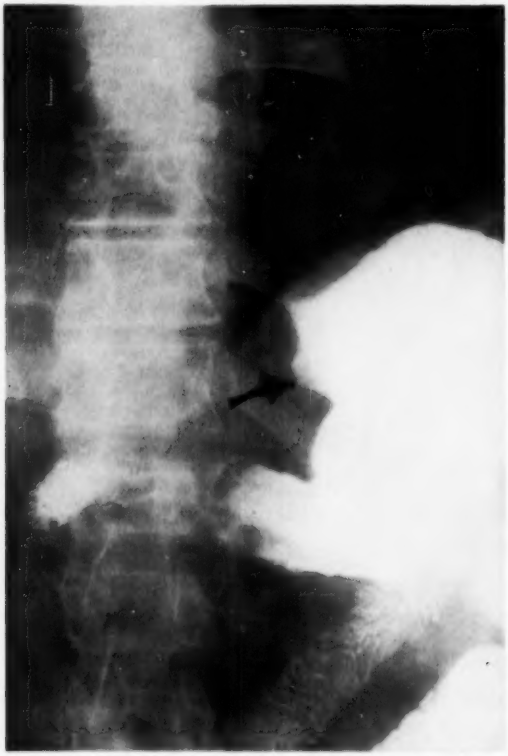

Fig. 9—Barium study of the stomach showing a penetrating gastric ulcer of lesser curvature. The patient is a 57-year old man who had one episode of hematemesis (Case 7). He has refused surgery and is now being treated medically.

She is under the care of a competent gastroenterologist. The possibility that carcinoma may develop in this patient has been considered and the family has been so informed.

Case 5:—This patient was a 46-year old housewife who had a severe gastric hemorrhage while in Europe. A subtotal gastrectomy was performed at that time. After residing in America for several months she experienced another episode of bleeding for which she was hospitalized. The patient was prepared for

surgery via multiple transfusions to combat shock and an exploratory laparotomy was performed. The findings at operation showed that the stomach and small intestine were filled with old and recent blood. Apparently the previous operation was an exclusion procedure because one-third of the distal stomach remained attached to the duodenum. This gastric segment was opened and a gush of blood emanated from the area. The blood was suctioned away revealing a large polyp in a duodenal diverticulum (first portion). The bleeding apparently arose from an ulcerated area on the polyp. The distal gastric remnant and first

Fig. 10

Fig. 11

Fig. 10—Roentgenogram of stomach of patient described as Case 8. The patient was a 65-year old man with proven carcinoma of the stomach. His history was of three weeks' duration with one episode of hematemesis. He died three days following a total gastrectomy.

Fig. 11—Gastric x-ray of a 52-year old man with initial hematemesis 6 months ago. He refused surgery. This x-ray was taken three weeks after his hemorrhage. In addition to a duodenal ulcer there is marked gastritis. This photograph shows diffuse gastritis throughout the entire stomach. Since this x-ray was taken the patient has had two more episodes of serious bleeding. He refuses surgery (Case 9).

portion of the duodenum were resected. The patient had a stormy postoperative course and expired on the sixth postoperative day.

Case 6:—A 50-year old man, who had no severe complaints excepting flatulence and "belching" after meals, was seen as a hospitalized patient. These symptoms were so frequent that he accepted them as part of his postprandial digestive process. His symptoms did not perturb him until he had an episode of hematemesis. Although this hemorrhage was not a severe one, he sought hospital care. After several weeks had passed a barium swallow demonstrated a pseudodiverticulum of the stomach due to a perforating gastric ulcer. The

patient was operated upon and a very high subtotal gastric resection performed for a benign gastric ulcer. He had an uneventful recovery.

Case 7:—A 57-year old laborer had no illnesses until he experienced one episode of hematemesis in October 1953. He was hospitalized and treated without surgery. Following a subsidence of symptoms a barium study of the upper gastrointestinal tract was performed. This revealed a duodenal deformity consistent with ulceration. The more interesting finding, however, was an ulcer high on the lesser curvature of the stomach. The patient has consistently refused surgery and is now under the guidance and care of a gastroenterologist.

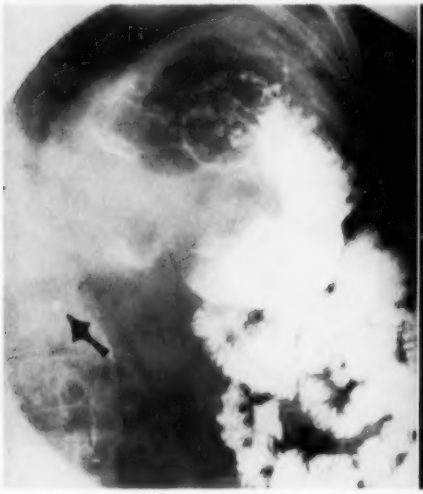

Fig. 12

Fig. 13

Fig. 12—Roentgenogram of the stomach of a 63-year old housewife with initial hematemesis. This x-ray shows a diverticulum in the second portion of the duodenum. Other x-rays demonstrated a duodenal ulcer and a hiatus hernia (Case 10).

Fig. 13—Barium studies of the stomach of the patient described as Case 11. This film demonstrates "stump" gastritis and inflammation at the site of anastomosis. This x-ray was taken three weeks following initial hematemesis and five years after a subtotal gastrectomy for an obstructing duodenal ulcer (Case 11).

Case 8:—A 65-year old man had a slight episode of hematemesis. He was treated at home by his family doctor. Three weeks later a barium study of his stomach was performed (cf. Fig. 10). This x-ray study revealed a gastric carcinoma. After an adequate preoperative preparation, he was operated upon. The distal half of the stomach was involved with carcinoma. A total gastrectomy was performed. He had a stormy postoperative course and expired on the third postoperative day.

Case 9:—A 52-year old auto salesman had an initial episode of hematemesis six months ago. He was hospitalized and surgery was advised at that time but

he refused. Barium studies performed six months after his initial hemorrhage demonstrated diffuse gastritis in addition to a duodenal ulcer. Since these x-rays were taken, the patient has had two episodes of bleeding which were not severe enough to be called a hemorrhage. In spite of all this bleeding he persistently refuses to be subjected to surgery.

Case 10:—A 63-year old housewife was hospitalized for initial hematemesis. In addition to the bleeding she had a simultaneous acute coronary occlusion. For this reason she was treated conservatively. Treatment consisted in three blood transfusions (500 c.c. each). Following the subsidence of the bleeding a barium study was made of her stomach. The x-rays demonstrated a hiatus

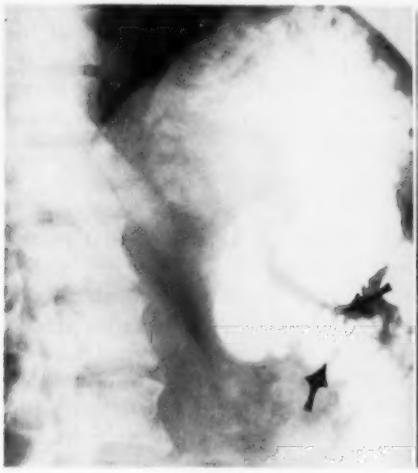

Fig. 14

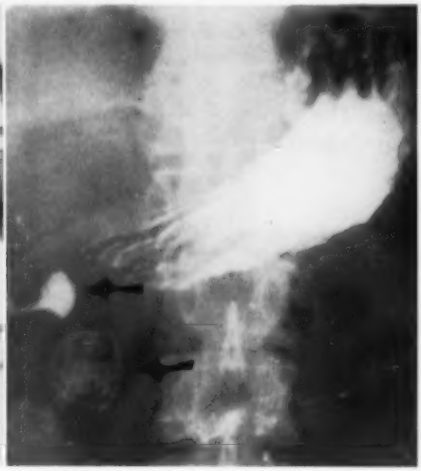

Fig. 15

Fig. 14—Barium studies of the jejunum of the same patient as described in figure 13. This film shows a "saw-tooth" pattern indicative of jejunitis. This jejunal involvement is associated with the gastritis visible in figure 13.

Fig. 15—Barium studies of the stomach and duodenum of the patient described as Case 12. This photograph shows two large duodenal diverticuli. The patient has refused surgery.

hernia, duodenal ulcer and a diverticulum in the second portion of the duodenum. Although the exact site of the hemorrhage is not definitely established because of the presence of several lesions, nevertheless this case study substantiates the primary purpose of this paper; namely, that initial hematemesis usually indicates the presence of serious underlying organic pathology. The patient was discharged from the hospital on a medical regimen.

Case 11:—A 55-year old barber was operated upon by me in 1948. At that time a subtotal gastrectomy was performed for an obstructing duodenal ulcer (no vagotomy). He was followed for two years after surgery and was free from any distress. He was not seen again until October 1953. His complaint at

that time was an initial hematemesis of moderate severity which subsided after two days. Since his operation his history is one of dietary indiscretion, heavy cigar smoking and moderate indulgence in alcoholic beverages. After the subsidence of the initial bleeding a barium swallow was administered. The roentgenograms revealed diffuse gastritis in the residual stomach (stump gastritis), inflammation at the anastomotic site and in the jejunum (jejunitis). At the present time the patient is being treated medically.

Case 12:—A 57-year old man was admitted to the hospital as an emergency patient because of hematemesis. His red blood count had fallen to 2,900,000 and his hemoglobin was 60 per cent. He was advised to have an operation but he refused to consent to surgery. He was therefore treated conservatively and responded well to therapy. Several weeks after he was discharged from the hospital a barium study was made of his upper gastrointestinal tract. These x-rays demonstrated two diverticuli in the descending portion of the duodenal horseshoe. The patient was discharged to his family physician who has reported that no further complaints have been in evidence.

Comment

Twelve instances are presented in which patients with an initial gastric hemorrhage of varying severity were found to have serious underlying gastric pathology. The pathology encountered ranged from duodenitis and gastritis to peptic ulcer and gastric carcinoma. Almost all of these patients had a similar history of questionable serious gastric complaints until the onset of hematemesis. These case reports indicate that one episode of gastric hemorrhage often indicates the presence of severe underlying gastric pathology. Such patients should be considered as surgical problems, with surgical intervention strongly suggested in those patients where there is indication for it. The surgical intervention may be immediate or temporarily delayed. Prolonged medical therapy for six to twelve months is not advocated because of the danger of a recurrent hemorrhage. Moreover, what is equally important the cause for the hematemesis may be serious organic disease, such as a perforating ulcer, severe gastroduodenitis or cancer.

Summary

1. Attention is called to initial gastric hemorrhage.

2. The cause for the hemorrhage may be serious organic gastric pathology as seen in most of the patients reported here.

3. Initial hematemesis is a surgical problem and conservative treatment as a prolonged regimen is not advocated because if the pathology is serious enough to initiate bleeding, it is serious enough to be removed.

4. It is preferable to operate too often for hemorrhage rather than to operate too late.

ROENTGENOLOGICAL STUDIES OF GASTRIC SECRETION IN CHILDREN

FRANZ J. LUST, M.D., F.A.C.G.*

New York, N. Y.

During the past years, the roentgenological examination of the upper gastrointestinal tract in children has found wide recognition, as demonstrated by several reports dealing with duodenal ulcers in children. All those interested in this field agree, however, that the roentgenological examination of children

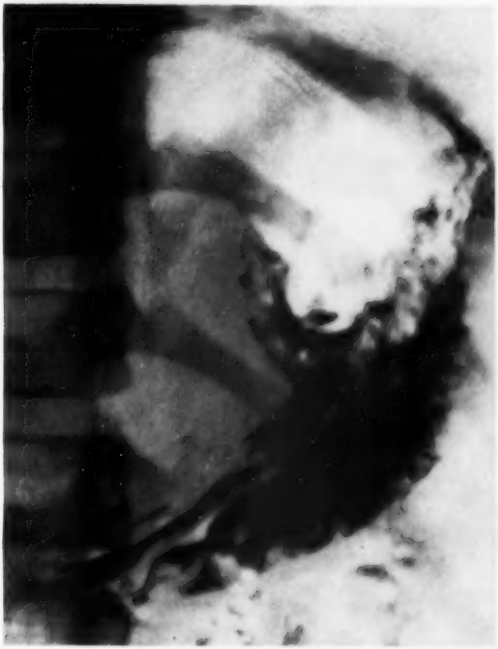

Fig. 1 —Normal mucosa of the stomach of a child 7 years old.

is difficult. We have evolved a new technic, which seems to be simple, thorough and efficient, and besides, has the advantage of relieving, as much as possible, the nervous stress and strain of our little patients.

Removal of Nervous Stress

It is of the greatest importance that the child should not be nervous or frightened before coming to the examination. This demands the greatest co-

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operation on the part of the parents and the referring physician. They should emphasize that the examination is not painful, that it will be performed in good bright light. A child who needs a roentgenological examination of this kind has not been sick for just one day, but probably has a prolonged history of illness and has become apprehensive. We are fortunate that the roentgenological examination of the upper gastrointestinal tract is performed without pain or

Fig. 2—Stomach in erect position. A normal amount of gastric secretion is present above the dense barium shadow. Child 8 years old.

discomfort. This fact should be made clear to the child and to the parents. The examination of children is different from that of adults.

Technic

The child drinks two ounces of diluted, chocolate-flavored barium suspension, then has to lie on a soft couch for a few minutes on each side. Only

now do we have the child lie on the roentgen table. A film in prone position shows the mucosal pattern of the stomach (Fig. 1). An advantage is that by this time, the pylorus has opened. The delayed opening, frequently met if the

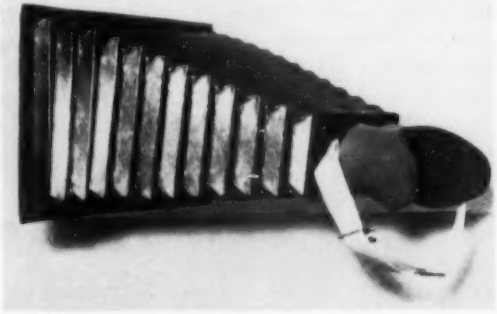

Fig. 3—Apparatus for fluoroscopy in normal light, shielding the eyes of the examiner. (Belloscope, Ampere X-Ray Co., New York City.)

examination is begun with fluoroscopy, is thus eliminated. This delay in the filling of the duodenum may be an entirely functional one, often due to anxiety.

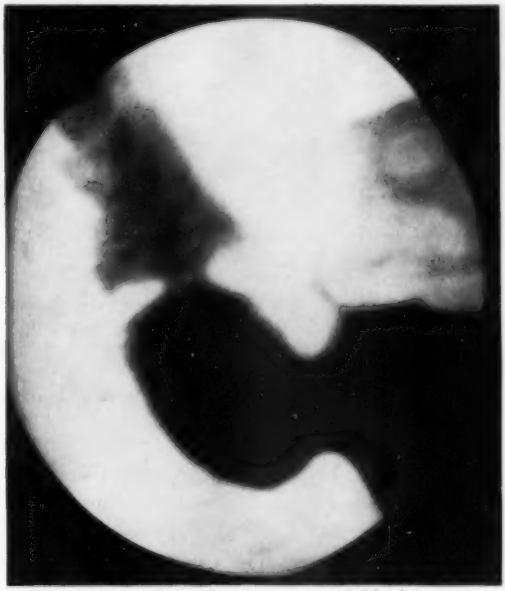

Fig. 4—Normal duodenal cap in a child of 8 years.

Another 3 ounces of barium is now administered and films are taken in prone, prone-oblique and erect positions (Fig. 2). The films are reviewed and the experienced physician sees most of the normal and abnormal features of the case.

Up to this time, the roentgen room can be kept in normal light. The examiner now uses a head fluoroscope, which, shielding his eyes against the light, enables him to fluoroscope the patient in bright light (Belloscope), (Fig. 3). The child is entirely unaware of what is going on. As the physician has seen the films, only a very short time for fluoroscopy is needed. A film of the stomach and small intestines ends the morning session and the patient is allowed to have breakfast. Two and six-hour films are taken. In case of the necessity for spot films (Fig. 4), these can be taken in red light which is not as frightening as complete darkness. Today's fluoroscopic screens are so well perfected that it is easy to localize the duodenal cap in slightly subdued light.

Studies of the Gastric Secretion

The secretory changes of the stomach can be studied by taking a gastric analysis. This procedure is not too frequently used in children as the swallowing

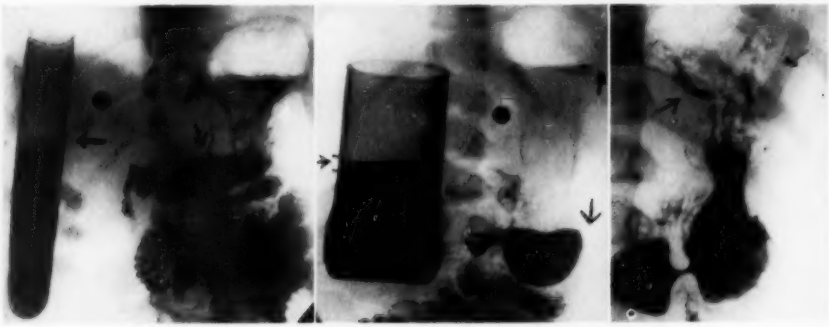

Fig. 5 Fig. 6 Fig. 7

Fig. 5—Increased gastric secretion seen above the dense barium shadow compared to the sedimentation in the test tube. Case of duodenal ulcer in a child, 7 years old.

Fig. 6—Increased gastric secretion compared to the small amount of sedimentation in the glass. Duodenal ulcer in a child, 7 years old.

Fig. 7—Roengenological appearance of mucus. Note the mottled appearance especially in the upper part of the stomach. Duodenal ulcer.

of a tube is not pleasant for them. Of course, this is the most accurate way. Roentgenologically, just by drinking a contrast substance, it is easier to study the amount of secretion. The empty stomach, normally, contains a very small amount of secretion. If we take a film in erect position approximately 20 minutes after administration of barium, at the end of the routine roentgenological examination we are able to see an area of diminished density above the barium shadow. We have already studied this phenomenon in adults and have found that it represents the secretion due to the stimulus of the ingested contrast substance.

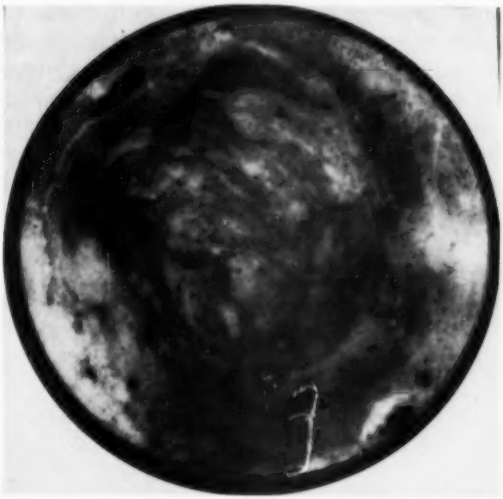

Fig. 8–Roentgenogram of mucus mixed with a barium suspension. Note the mottled appearance, as the mucus does not mix homogenously with the contrast substance.

In one of our tests, at the beginning of the examination (Fig. 5), we poured some of the routine barium suspension into a test tube. Twenty minutes later,

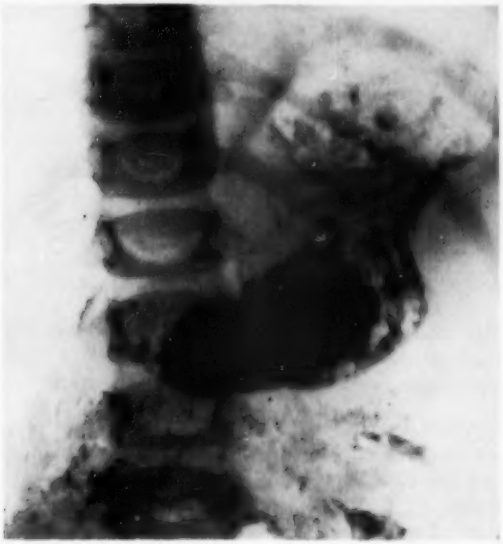

Fig. 9–Mucus in a 4-year old child with gastric distress after oral administration of antibiotics. Note the mottled appearance.

a film taken in erect position, showed the barium in the test tube to the left of the stomach. There was within a layer of diminished density above the barium, representing the sedimentation of the contrast substance. The stomach, however, showed a broad layer of diminished density above the barium. This represents the amount of gastric secretion within 20 minutes. A different test was performed, as the small amount of sedimentation in the test tube might have been due to the adhesive forces of the narrow tube. This time, a normal glass was used (Fig. 6), which had approximately the diameter of the stomach. Here again, the sedimentation inside the glass reproduced on the

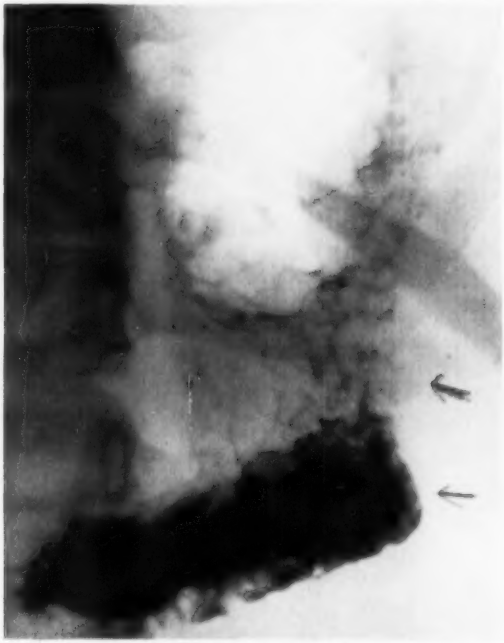

Fig. 10—Mucus in the stomach in a case of gastric distress in the presence of a chronic postnasal drip. Note the mottled appearance.

same film with the stomach, showed a small amount of sedimentation. In the stomach of this seven-year old patient, a heavy layer of secretion above the contrast substance is visible.

This is a very simple method to measure the amount of gastric secretion. The method is not disagreeable, even to difficult patients, and to a large extent, is able to supplant the use of a gastric tube. These studies give results similar to those on adult patients previously published.

The second part of the examination is the study of the gastric mucosa. Normally, the folds are readily outlined. The rugae are of even size and caliber,

reaching from the cardia to the pylorus (Fig. 1). They are slightly tortuous and clean-cut. In pathological cases, the folds can be seen, but the contrast substance shows small circular shadows. In an adult, we took some gastric contents, which contained a large amount of mucus. We tried to mix the mucus with barium and took a roentgenogram (Fig. 8). This showed that the barium did not mix homogenously with the mucus, but produced the same mottling effect as seen in mucosal studies. Therefore, it seems that the mottling is due to the presence of mucus in the stomach. Increased amounts of visible mucus seems frequently to be associated with some form of gastric hypersecretion. In cases of gastric distress after ingestion of antibiotics (Fig. 9) and also in the presence of postnasal drip, similar roentgen findings were observed (Fig. 10). No other signs of pathology were found and it was concluded that the presence of increased visible mucus was the cause of the gastric distress.

Conclusion

A new technic for the roentgenological examination of the gastrointestinal tract of children has been presented. The technic starts with the assurance to patients and parents that no pain or stress in involved. The technic primarily relies upon roentgenograms taken in different positions and the reduction of fluoroscopy to a bare minimum. Fluoroscopy is performed in normal light with the physician using an eye-shielding apparatus (Belloscope, Ampere Co.). The child is entirely unaware and unapprehensive of this procedure. The use of spot films is stressed in order to study the very rapid motility of the duodenum in children.

This simple roentgenographic method enables us to study not only morphology, but also function, such as secretion, and gastric mucus production.

Increased amounts of gastric secretion is a well-known factor in peptic ulcer and preulcer conditions. Therefore, if increased amounts of gastric secretion are found, a restudy may eventually demonstrate the presence of an ulcer. We should like to draw the attention of the pediatrician to the observation and demonstration of increased, visible, gastric mucus. This phenomenon was observed in cases of ulcer, postnasal drip and after oral administration of antibiotics.

Only prolonged clinical studies will reveal the significance of increased gastric mucus. Functional studies of the gastrointestinal tract are of the greatest importance in children, as functional changes tend to develop into organic pathology in later life.

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GASTROSCOPIC VISUALIZATION OF THE LESSER CURVATURE OF THE STOMACH WITH THE NEW ROTATING TABLE

COLEMAN C. JOHNSTON, M.D.

Lexington, Ky.

Because of our interest in the lesser curvature of the stomach we, as gastroscopist, have suffered many frustrations. At least in our own experience part of this pathway has until recently fallen into the category of a blind area. For this reason, it would seem practical to review briefly an experience which, for us, has rolled back this curtain to expand our horizon so that, with almost regularity, we are able to follow the entire course of the lesser curvature, as well as visualize much of the upper posterior wall.

By means of a rotating table designed and built under our direction by Dr. Karl Lange, Professor of Aeronautic Engineering at the University of

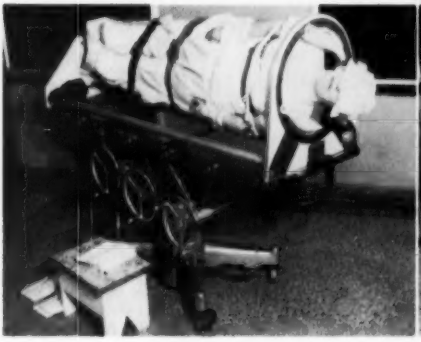

Fig. 1

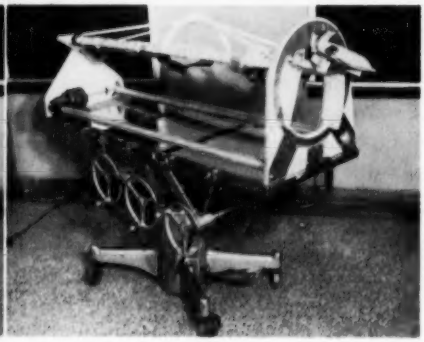

Fig. 2

Fig. 1—The patient with head extended is fixed in place on the rotating gastroscopic table. She is facing the ten o'clock position. The nine o'clock position corresponds to the conventional left lateral position ordinarily used in gastroscopy.

Fig. 2—The rotating gastroscopic table is shown facing the six o'clock position. The 1/20th horsepower motor controlled by a double circuit switch is well able to rotate a 230 pound man.

Kentucky, we have through persistence and luck, stumbled on to this interesting technic. As seen in the accompanying illustration, a patient reclines on the rotating table and is fixed in place with "aviation seat belts" over the chest, pelvis and ankles. The patient is then rotated "spit-like" from the supine position, clockwise around to the left lateral position, routinely used for gastroscopy. This maneuver gives the patient a trial run and the examiner an opportunity to see that all is in order. The gastroscope is then introduced and the patient quickly examined in this position. By means of a double circuit switch controlling a one-tenth horsepower electric motor, one may rotate the patient in

either a clockwise or anticlockwise direction and stop at any desired point in the 360 degree circumference.

In our early experience we fell into the routine of clockwise rotating arresting the table at twelve, three and six o'clock, but also observing during the rotation. With time it occurred to us that the lower third of the lesser curvature seemed to fall away from the gastroscope as rotation progressed from the twelve toward the three o'clock position. As the force of gravity still further drew the stomach downward with the patient moving through the four, five and six o'clock positions, the middle and lower thirds of the lesser curvature of the stomach always presented at some point during the rotation. Encouraged with these observations, though at first skeptical of our findings, it became essential to prove their certainty.

It occurred to us that if a Miller-Abbott tube was introduced the day before examination and the tip visualized with the fluoroscope to be well down in the duodenum, then it could be fixed by inflating the balloon. With traction applied the tube could then do nothing more than hug the lesser curvature, the shortest distance between the duodenum and the esophagus. At gastroscopic examination with the Miller-Abbott tube thus anchored and the patient on the rotating gastroscopic table, we were able to follow the tube with ease from the pylorus along the entire course of the lesser curvature and see it actually enter the esophagus. The greater part of this gratifying experience occurred during the rotation between the two and six o'clock position. Incidentally, we have also observed that at about five o'clock, the pylorus can, in our experience, be seen in its entirety far more often than in the conventional left lateral position, also more of the posterior wall is seen.

On withdrawing the Miller-Abbott tube from the duodenum during this study, the balloon was deflated and when the thought occurred to us that the withdrawal should have been made with the balloon inflated, it could not be re-introduced.

In view of the difficulty encountered in establishing the diagnosis of the channel ulcer, this maneuver would seem of practical interest.

The possibility of evaginating proximal duodenal mucosa, presents further fascinating fields for consideration. Could the early anterior, superior or inferior wall duodenal ulcer be evaginated into view. Could any of these or even the old fixed posterior wall ulcer be seen through an inflated transparent balloon?

We have not as yet had the opportunity to further explore these intriguing possibilities.

President's Message

Our Regional Meeting in Memphis is now history, and marks another milestone of accomplishment for our College. Credit goes to Dr. John E. Cox and Dr. E. G. Campbell for their splendid management, and to the excellent essayists, who contributed so much to make it possible.

The various committees and the Trustees gathered a little early for informal discussions, in order to expedite the regular meeting of the Board. Dues, fees, and credentials were carefully reconsidered, and schedules set up for the review and approval of the Fellows at the Chicago meeting. You will receive notes on the action of your Board of Trustees very soon and, it is hoped, you will like them.

Dr. Kirchner and his committee have now completed the program for the Chicago meeting. It has been reviewed, and unanimously accepted by the Board.

Dr. Wirts and his committee still have a few details to perfect on the Postgraduate Course, which in turn promises to be one of the best we have ever had. I hope you will take this occasion to block out the time to include the course in your Chicago sojourn and, I hope, you will induce your friends to do the same. The discussions by Drs. Wangensteen and Snapper following each speaker, are something you cannot afford to miss. The sound opinions of these men, is basic to our effort.

It is obvious that our College is growing stronger, and that our membership at large is catching the keynote of successful production. We must evaluate progress at intervals, and this must include individuals in responsible positions. Their advice and judgment must temper our every action. On that basis, we can always perpetuate our committees with experienced men, and our objectives will be better attained.

Our College needs workers in the younger age brackets, men with ideas, and the fire to develop them. Let us know who and where you are.

Lyon A. Terry, M.D.

NEWS NOTES

Dr. Samuel Weiss Receives Plaque

Samuel Weiss, M.D., F.A.C.G., Editor-in-Chief of The American Journal of Gastroenterology since its inception in 1934, was signally honored by the New York Academy of Gastroenterology on the occasion of his 70th birthday.

At the Annual Meeting of the New York Academy of Gastroenterology, the New York affiliate of the American College of Gastroenterology, held on 16 May 1955, Dr. Weiss was presented with an illuminated plaque. The plaque, which contained a set of resolutions recognizing Dr. Weiss' 70th birthday and his accomplishments and achievements in medicine generally and in the American College of Gastroenterology specifically, was signed by Frederic W. Bancroft, M.D., F.A.C.G., outgoing President; Hyman M. Robinson, M.D., F.A.C.G. outgoing Secretary and William C. Jacobson, M.D., F.A.C.G., Treasurer of the New York Academy of Gastroenterology.

Dr. Jacobson presented the award to Dr. Weiss on behalf of the officers and members and praised Dr. Weiss' accomplishments in glowing terms. Over 50 members of the Academy were present at the Annual Meeting.

Officers elected to serve for 1955-1956 are: President, Franz J. Lust, M.D., F.A.C.G.; 1st Vice-President, A. X. Rossien, M.D., F.A.C.G.; 2nd Vice-President, Jerome A. Marks, M.D., F.A.C.G.; 3rd Vice President, Milton J. Matnzer, M.D., F.A.C.G.; Recording Secretary, Alexander Slanger, M.D., F.A.C.G.; Corresponding Secretary, Joseph Roger Van Dyne, M.D., F.A.C.G. and Treasurer, William C. Jacobson, M.D., F.A.C.G.

Flowers were presented to Mrs. Weiss on behalf of the Academy as a token of appreciation of her graciousness as hostess at the Annual Meeting held in Dr. and Mrs. Weiss' home.

The good wishes of Lynn A. Ferguson, M.D., F.A.C.G., President of the American College of Gastroenterology and Mrs. Ferguson were extended to Dr. Weiss in the form of a dozen red roses. Other members of the New York Academy of Gastroenterology expressed their good wishes in telegrams.

Ladies Auxiliary News

Mrs. Lynn A. Ferguson, President, Mrs. Elihu Katz, Secretary, Mrs. Joseph Shaiken, Chairman of the Program Committee and Mrs. Samuel Weiss, all of the Ladies Auxiliary of the American College of Gastroenterology, met in Memphis, Tenn., on Sunday, 24 April 1955 to discuss plans for the ladies activities at the forthcoming Second Annual Convention of the American College of Gastroenterology to be held in Chicago, Ill., this coming October.

The tentatively arranged program includes a business meeting on Sunday afternoon, 23 October; attendance at the convocation ceremony of the College; a tour of the Museum of Science and Industry; a sightseeing tour of Chicago; a tour of the world-famous Merchandise Mart; participation in the Annual Dinner-Dance of the College and a luncheon and fashion show at the fabulous Marshall Fields department store.

Complete details of this exciting program will be sent to the ladies who are members of the Ladies Auxiliary in a letter now being prepared. While all ladies are invited to participate, the auxiliary would welcome those who wish to join now. Dues are $2.00 a year and they may be sent to the Treasurer, Mrs. Louis L. Perkel, 131 Kensington Ave., Jersey City, N. J.

New Fellowship Keys

New Fellowship keys for Fellows of the American College of Gastroenterology have been authorized by the Board of Trustees. An illustration of the new key is at the left.

Those who were elected or advanced to Fellowship in the American College of Gastroenterology since the Annual Meeting of the College in October of 1954, will receive the key without additional charge at the convocation ceremony in Chicago on Sunday, 23 October 1955.

Other Fellows may order the new keys from the headquarters office, 33 West 60th Street, New York 23, N. Y., at $10.00 each including federal tax and shipping charges.

The reverse side of the key will be engraved with the name and date of election to Fellowship. Send your orders in today.

In Memoriam

We record with profound sorrow the passing of Dr. Philip Ladin, Associate Fellow, of New York, N. Y. We extend our deepest sympathies to the bereaved family.

ABSTRACTS FOR GASTROENTEROLOGISTS

GASTROINTESTINAL TRACT

HEMORRHAGE FROM THE UPPER GASTROINTESTINAL TRACT: H. M. Pollard. Illinois M. J. 107:1 (Jan.), 1955.

Proper management of upper gastrointestinal hemorrhage requires the establishment at an early moment of the approximate amount of blood loss and the source of the hemorrhage. From these the most effective course of treatment then becomes a matter of clinical judgment and experience. Special attention must be paid to the bleeding patient with sclerotic vessels.

Mild to moderate hemorrhage may be treated with simple conservative methods that carry almost no mortality. The author prefers barbiturates for sedation and avoids morphine. He believes that prompt feeding is highly desirable and uses the Meulengracht regime with added antacid and antispasmodics. Transfusions are, of course, valuable but there is reason to believe that massive rapid transfusion is neither indicated nor desirable and small transfusions by the drip technic are better. Effective surgical treatment must be undertaken during the first 24-48 hours after the onset of therapy and is particularly indicated in patients over the age of 60 where there is evidence of either continuous bleeding or recurrence of bleeding.

LOUIS A. ROSENBLUM

MASSIVE UPPER GASTROINTESTINAL HEMORRHAGE: ROLE OF EMERGENCY SURGERY IN MANAGEMENT: Evan W. Schear, Warner F. Bowers and Benj. H. Sullivan. Military Med. 116:109 (Feb.), 1955.

Patients with severe upper gastrointestinal hemorrhage seem to fall into 3 groups: (1) Those who continue to bleed rapidly (2) Those who bleed intermittently, and (3) Those who stop bleeding soon after onset and recover quickly.

The criteria of continuous bleeding are the loss of plasma fluid with development of shock and the drop in hemoglobin and red blood cells.

The authors recommend an aggressive approach to the first two groups, specifically: adequate and rapid blood replacement, definitive diagnostic assay such as barium x-ray study, esophagoscopy if varices are suspected and a decision to be made whether it would not be dangerous to allow the patient to go without surgery.

Prompt exploration within 48 hours, with subtotal gastrectomy as the choice operation, should be done if bleeding continues or recurs having once stopped.

H. M. ROBINSON

LEFT PARADUODENAL HERNIA: H. O. L. Murray. Canad. M.A.J. 72:263 (15 Feb.), 1955.

This case report deals with a left paraduodenal hernia. Intraabdominal herniae involving the duodenal fossae are rarely diagnosed clinically. Left paraduodenal hernia is hernia into the descending mesocolon.

The fossae in this region have been described by Treitz, Lord Moynihan, Waldeyer and others.

In this instance, the patient had undergone an operation four and one-half years previously for the closure of a perforated

duodenal ulcer and "the diagnostic impression was that of small bowel obstruction, probably from postoperative adhesions."

The author makes this observation, that the abdominal distention was definitely more marked on the left side: and in the previous work-up, the barium meal had not been followed into the region of the ligament of Treitz. In addition, he points out that x-ray examination of the abdomen both in the supine and erect positions, is of great value in that there is limited mobility of the small bowel loops.

Lord Moynihan's description of the fossa is mentioned by the author as is the constant relationship of the inferior mesenteric vein: bowlike, superior, anterior and to the left of the hernial mass.

IRVIN DEUTSCH

ESOPHAGUS

CARDIOSPASM: John E. Summers. Am. Pract. & Digest. Treat. 6:44 (Jan.), 1955.

Cardiospasm is a nonorganic stenosis of the lowermost portion of the esophagus in which a coordinated distally travelling peristaltic wave is absent. The cause is thought to be an inflammatory reaction in Auerbach's plexus with a loss of ganglion cells and replacement by interstitial supporting cells. Thus a preponderance of sympathetic control occurs, contracting the circular fibers of the terminal 2-3 cm. of the esophagus causing stenosis at the site with secondary marked dilatation above.

Treatment suggested: 80-90 per cent of the cases can be successfully treated by dilatation.

A 41 F sound is passed over a previously swallowed silk thread. This is followed by a second dilatation with a 50 or 60 F Plummer sound. In 48-72 hours subsequent dilatations are carried out by a hydrostatic dilator with water pressure of 22-24 feet, maintained for a few seconds. If complete relief is not obtained a second hydrostatic dilatation can be performed in 48-72 hours after the first. Two or three dilatations are usually sufficient for successful treatment. The success of dilatation depends upon the rupture of the constricting circular muscle fibers at the stenosing site. This treatment has its dangers. Rupture of the esophagus, mediastinitis, hemorrhage and aspiration pneumonitis are not uncommon.

Should dilatation fail, early surgery is urged. The majority of thoracic surgeons now prefer the Heller Type of operation which consists of reaching the cardia through a left thoracotomy. An incision is made in the esophageal musculature and the mucosa is allowed to bulge through this opening relieving the obstruction.

A. J. BRENNER

AN X-RAY AID TO THE DIAGNOSIS OF RADIOOPAQUE FOREIGN BODIES IN THE UPPER INTRATHORACIC ESOPHAGUS: B. S. Epstein. Am. J. Roentgenol. 73:115, (Jan.), 1955.

The authors present technics for use where there is a suspected foreign body in the uppermost portion of the intrathoracic or lower cervical esophagus. They take routine lateral roentgenograms of the neck. If the suspected object is found, the study is discontinued. If however, nothing is evident on this film they then obtain lateral neck roentgenograms at the height of deglutition. This moves the laryngeal cartilages sufficiently out of the way to make the object visible.

J. R. VAN DYNE

DISEASES OF THE ESOPHAGUS: G. V. Brindley, Jr. Texas J. Med. 51:57 (Feb.), 1955.

Chemotherapy and improved anesthesia have changed the surgical technics for diseases of the esophagus, the large pulsion-type diverticula at the cricopharyngeal level and those of the lower third of the esophagus formerly corrected by a two-stage operation can now safely be done in one step. The traction type of the mid-esophagus

usually requires no surgical intervention.

Atresia of the esophagus with absence of gas in the stomach, indicating complete obliteration of the lumen at some level, mitigates against a good primary anastomosis, the tracheo-esophageal fistulous type, approached by a right extrapleural or transpleural route assures good primary anastomosis, with ligation of the fistula.

In achalasis, section of the lower esophageal muscularis followed by dilatation usually suffices.

Acquired strictures as a rule are amenable to dilatation, occasionally an esophagogastrostomy, or a Roux esophagojejunostomy is required.

Benign tumors occur rarely and are treated by simple dissection.

Spontaneous rupture of the esophagus can be handled as a simple wound with debridement, closure of the tear and liberal antibiotic therapy.

Cervical cancer has four methods of operation; resection of the lesion and reconnection with a skin-lined tube; block dissection of the larynx, cervical esophagus and lymph nodes and substitution of a skin-lined tube; resection of the lesion and a high esophagogastrostomy; simple resection esophageal anastomosis.

Replacement of a mid-thoracic cancerous section of esophagus with a plastic tube has been used since 1952 after the technic of Berman.

Cancer of the lower esophagus usually involves the cardia and a left transthoracic gastrectomy with low esophagectomy, followed by esophagogastrostomy is the operation of choice.

All cancers should receive irradiation therapy after extirpation.

J. Edward Brown

STOMACH

ROENTGEN APPEARANCE OF GASTRIC INVASION FROM CARCINOMA OF THE COLON: Arnold L. Bachman. Radiology. 63:814-822 (Dec.), 1954.

This article reports four cases of colonic carcinoma invading the stomach. It again points out the necessity for studying the structures contiguous to the greater curvature of the stomach in those instances where filling defects are found.

The salient feature of these extragastric neoplasms is that they are in essence intramural tumors. The demonstration, however, of a fistulous tract is a significant factor against the diagnosis of primary gastric intramural growth.

It is of interest to note that three of the four cases reported showed no evidences of lymph node, liver or distant metastases at the time of operation, despite the extension of the colonic growth into the stomach.

Irvin Deutsch

EXFOLIATIVE CYTOLOGY: C. E. Rubin, M. I. Klayman and J. B. Kirsner. M. Clin. North America pp. 261-280 (Jan.), 1955.

The study of exfoliative cytology is a valuable method for the diagnosis of gastrointestinal cancer, which now accounts for 40 per cent of cancer deaths in the U. S. This method is considered to be 85-90 per cent accurate when interpreted by adequately trained personnel.

Esophagus:—The collection of cells from the esophagus is relatively simple. A Levin tube is passed to the level of the lesion and after several minutes of lavage with Ringer's solution yields a satisfactory specimen. It is usually unnecessary to pass a stenotic lesion. The specimen should be fixed at the bedside to avoid digestion. This method is especially valuable when esophagoscopy or biopsy is negative or indeterminate. The ease of esophageal lavage justifies its more frequent use but the interpretation of the slides must be the responsibility of the trained cytologist.

Stomach:—Preparation is important in gastric cytological studies: either an overnight fast or lavage followed by suction in the case of obstruction. The methods employed are simple saline lavage, an abrasive balloon, and a special antral abrasive balloon with a mercury weight. In order to make available the cells in the mucus covering of the gastric mucosa, the mucolytic

enzymes papain and chymotrypsin have been employed. The material must be fixed as soon as possible because of the digestive properties of gastric juice. Cytological studies are indicated when the diagnosis by x-ray and gastroscopy remains uncertain, especially in areas such as the cardia and the antrum. An exploratory operation may be avoided, with radiologic and endoscopic examinations repeated to establish the diagnosis. No single method is infallible and exfoliative cytology will not help when the lesion is intramural or covered with a necrotic exudate. Gastric cytology is especially helpful in differentiating a benign from a malignant gastric ulcer. Also by this means a lymphoma may be detected preoperatively.

Duodenum:—These studies are a welcome aid in diagnosing malignancies of the biliary tract, pancreas and duodenum. The tube is passed into the duodenum. Drugs may be given to increase the pancreatic and biliary secretions. The collection vessels should be packed in ice to retard digestion of the specimen.

Colon:—Lesions of the descending colon and sigmoid are diagnosed more accurately and easily than those of the ascending and transverse portions of the colon. The colon is prepared as for barium enema or sigmoidoscopy and about a liter of saline or Ringer's solution is used to lavage the area. Cytological examination may be used when the diagnosis is in doubt by x-ray, and may correctly indicate a benign lesion when other procedures suggest malignancy. In conditions such as regional enteritis or diverticulitis where x-ray suggests possible malignancy, cytological study may be the decisive factor in arriving at the correct diagnosis.

ARNOLD STANTON

LIVER AND BILIARY TRACT

THE PROBLEM OF BILIARY DISINFECTION: Michele Demole. Therap. Umschau 5:17-22, 1953.

The problem of biliary disinfection has not been solved and the new antibiotics which are revolutionary in particular domains have not assisted in solving this problem, because the anatomic-physiologic conditions are unfavorable. Rendering bile sterile is relatively easy because disinfectants reach it without difficulty. However, bile is continually reinfected in traversing the passages, as well as within the gallbladder where it undergoes concentration. The walls of the gallbladder are refractory to sterilization through medication.

Attempts at obtaining disinfection by chemical methods continue in lieu of anything better. Included among these are: the iodides, methylene blue, menthol, acridine derivatives such as trykaflavin, etc. Demole states that he has discontinued using these drugs but that salicylates and urotropine (hexamethylenetetramine) still are widely utilized.

For the past two years the author has employed a biliary disinfectant introduced commercially as *Bilamid*. It is the oxymethyl-amide of pyridine-3-carbonic acid. This compound disintegrates in the body forming an aldehyde. In aqueous solution this aldehyde further decomposes to produce formaldehyde. The hydrogen ions, liberated by this reduction reaction, unite with the proteins within the bacterial molecules and serve to inactivate it. By so doing this medication acts as an antibacterial agent.

The author states that Bilamid should always be prescribed in large dosage (2 tablets of 0.50 grams each, three times a day for a total of 3 grams) over a relatively short period (five to eight days) but should again be restarted after a one week lay off; a schedule which should be followed for a minimum of three months.

Demole summarizes his detailed discussion by stating: The problem of biliary disinfection is complicated by anatomical and physiological difficulties and the futility of rendering the bile sterile, due to perpetual reinfection by residual germs in the walls of the bile passages; poor vascularization of the gallbladder except during attack of acute inflammation. Acute conditions require a simultaneous attack upon the focal infection located in the intestinal canal. Antibiotics do not provide a solution for the problem.

The modern method consists of combining a cholagogue with a disinfectant capable

of establishing a high concentration in the biliary passages. The compound Bilamid combines the antiseptic of formaldehyde with cholagoguic properties of nicotylamide producing cellular protection.

Clinical experience has proved that Bilamid is the present elective biliary disinfectant. The therapeutic results have been satisfactory in localized infections such as: cholecystitis, cholangitis, etc. The colon bacillus, which is the usual responsible agent, is particularly susceptible to attack from this medication. Bilamid is notable for its rapid onset but is of relatively short duration.

REGINALD B. WEILER

ACUTE CHOLECYSTITIS IN CHILDREN: C. C. Hopmans. Canad. M.A.J. 72:127 (15 Jan.), 1955.

The author reports a case of acute cholecystitis in a 6-year old boy, who developed crampy periumbilical pain, which later localized in the right upper quadrant, accompanied by moderate elevation of temperature and nausea but no vomiting, with marked tenderness in the right upper quadrant. Chest x-ray showed pneumonitis involving the right lower lobe. He was placed on antibiotics but when the temperature and white count rose laparotomy was performed. The findings were an acutely inflamed gallbladder with areas of necrosis but no stones.

Acute cholecystitis in children, although uncommon, must be kept in mind when evaluating abdominal pain. More frequent is acute appendicitis, especially if the appendix is high and retrocecal, and the recently stressed peptic ulcer.

ARNOLD STANTON

INTRAVENOUS CHOLANGIOGRAPHY: L. Potvin and J. P. Dugal. Canad. M.A.J. 72:90 (15 Jan.), 1955.

The authors discuss the uses of intravenous cholangiography with the aid of Cholografin. They report 19 patients, 8 of whom had had cholecystectomy, with visualization of the bile ducts in 13 cases (about two-thirds). Stones in the common duct were found in three cases and confirmed at operation. Cholografin showed calculi in a gallbladder which had appeared normal on two occasions by oral means, and calculi in a second gallbladder which had twice failed to visualize.

The patient must be tested for sensitivity before using Cholografin and it must be injected slowly, otherwise restlessness and nausea occur. Cholografin should be employed when the gallbladder is not visualized by oral means or when the oral procedure shows a normal gallbladder when there are positive clinical signs of biliary disease. Its main indication is for the visualization of the biliary ducts.

ARNOLD STANTON

PANCREAS

CYSTIC FIBROSIS OF THE PANCREAS ECCHINOSIS: B. M. Kagan. Illinois M. J. 107:3 (March), 1955.

Cystic fibrosis of the pancreas is a lesion involving the pancreas, broncheal, salivary and sweat glands. Disease is congenital and hereditary. It is progressive, causing partial and later complete pancreatic duct obstruction. Prognosis is good for immediate future but poor for long term. Symptomatology is varied.

Treatment—high calorie, high carbohydrate and protein, low fat with frequent meals. Infants do well on casein hydrolysate or dried skimmed milk powder with carbohydrates. Vitamin A, D, and K in aqueous with A and D twice normal dose. Antibiotic or sulfonamides depending on identifying organisms, sensitivity and response. Prophylactically all these children should be given a broad spectrum antibiotic such as tetracycline. Replacement therapy should consist of Viokase or Panteric granules. The psychological aspect should not be neglected. Occasionally antihistamines, bile salts and expectorants may be helpful.

LOUIS K. MORGANSTEIN

PANCREATOGRAPHY: H. Doubilet, M. H. Poppel and J. H. Mulholland. Radiology 64:325-339 (March), 1955.

This article is a most interesting study based upon 100 patients who were examined as follows: this procedure was performed at operation or in the postoperative period and consisted of the injection of a radio-opaque (Diodrast) through a plastic tube that had been inserted into the main pancreatic duct.

By this method, a) obstructive lesions of the pancreatic duct produced by calculi or carcinoma are well demonstrated: b) the origin, spread, persistence and treatment of pseudocysts can be followed: c) pathological changes seen in pancreatitis can be studied. In 39 per cent of all cases, the accessory duct is well demonstrated.

The authors have shown that 70 per cent diodrast produces active trypsin response rather than inactive trypsinogen and that the entire pancreatic acinar parenchyma may be visualized due to increased permeability as a result of the inflammatory reaction. It is possible, therefore, to opacify the entire pancreas and by this method to facilitate the search for adenomas; since adenomas are not connected with the duct system, the filling defect or negative shadow would l... hyperins...

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2. Riese, J. A.: Am. J. Digest. Dis. *21*:81, 1954.

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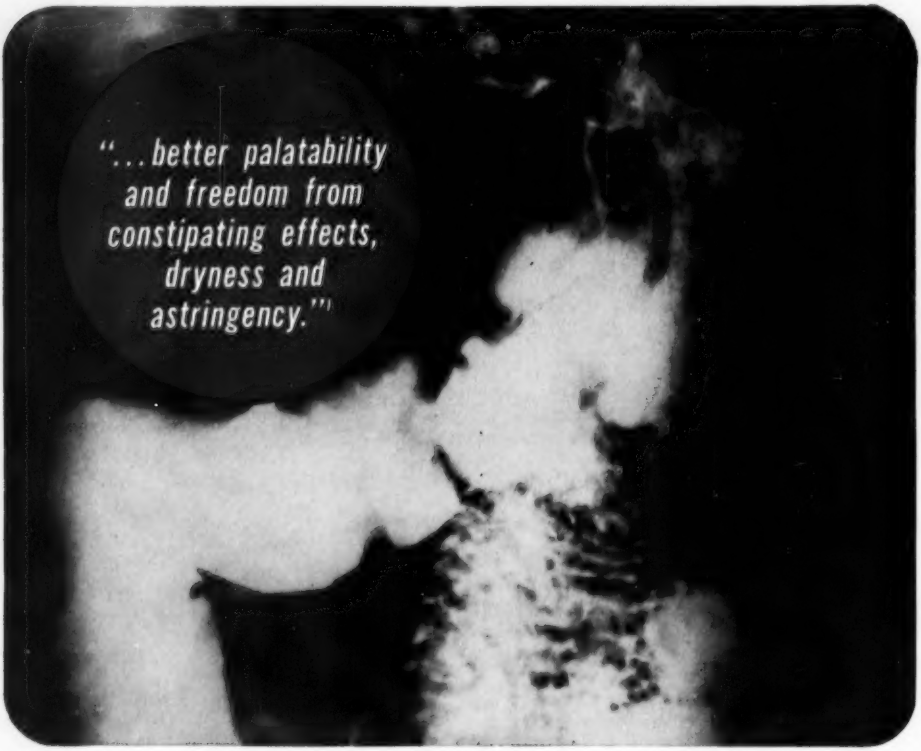

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1. Morrison, Samuel: Magnesium aluminum hydroxide gel in the antacid therapy of peptic ulcer, Am. J. of Gastroenterology 22:309 (Oct.) 1954.

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